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IX.

SURGERY OF THE FACIAL NERVE.

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Aside from the report of five cases of facial hypoglossal anastomosis for the cure of permanent facial paralysis that I have performed, and the demonstration of a gross specimen removed postmortem from one of these cases which had been cured for over a year, showing the perfect union of the two nerves, I have nothing original to offer in this line of work. Consequently the large part of my article is a repetition of what is already well known and on record. I have, however, written up this subject with one special purpose in view, namely: Inasmuch as the greatest number of facial paralyses are due to pathologic conditions within the temporal bone, more especially in the field of the otologist, or following his operative procedures to eradicate it, as, for instance, the radical mastoid, jugular bulb and labyrinthine operations, I have decided to bring this subject before you *in extenso*, compiling all that is practically known of the anatomy, physiology, pathology and treatment of the facial nerve.

Up to the present time, this subject is considered more particularly in general surgery, neurology, and internal medicine, but it evidently falls within the realm of the modern otologist.

It becomes frequently necessary to do a radical mastoid operation, in order to cure a facial paralysis, or if following such an operation one obtains a permanent facial paralysis, there is every reason why the otologist should correct this condition and not depend on a general surgeon to do so.

While the operation is very delicate and tedious, it is not difficult, and certainly the delicacy and tediousness of any operation are more characteristic of the work of the otologist than that of the general surgeon.

The operations for the cure of facial paralysis have gained considerable favor in the past five years, so that at the present time we have a great deal of material at our disposal to study the results from various methods. Murphy's article on surgery of the nervous system compiles practically all the literature up to the present day, comprising thirty-four cases, and completely goes over the pathology of the peripheral nerves. I am, through the kindness of Dr. J. B. Murphy, using that part of his article practically verbatim.

In order to study the cure of facial paralysis, one should have a clear knowledge of the anatomy and function of the facial nerve, with all its associated branches. I have, therefore, prepared these cuts, which will also serve to show the relationship in studying the anastomosis of the various nerves with the facial. Figure 1 shows the course of the facial nerve from the beginning at the cortex (upper segment), traversing the internal capsule and associating or uniting with the nucleus of the facial nerve at the floor of the fourth ventricle. From thence we speak of the facial nerve proper, or the lower segment. The fibers, then, pass in a curve about the nucleus of the sixth nerve, making what is known as the knee, being then joined by the eighth or auditory nerve, as two distinct trunks. These nerves have their exit from the brain at the junction of the pons and medulla. A third little nerve, known as the nerve of Wrisberg, or nervus intermediaris, is seen between these two trunks in their course towards the internal auditory meatus. At this latter point the two nerves are in a distinct relation; the auditory is posterior to the facial. They then pass into the internal auditory meatus until the aqueductus Fallopii is reached, when the facial leaves the auditory nerve

and passes into this (Fallopian canal) across the roof of the middle ear cavity. Then it takes a turn backwards and downwards, so as to occupy the innermost part of the lower half of the posterior meatal wall; it then passes through the bony canal in the mastoid process until it reaches the stylo-mastoid foramen. Here it again takes a horizontal turn towards the posterior surface of the parotid gland, just below the cartilaginous external auditory meatus, piercing the capsule of this gland, and entering the substance of it.

The nerve then divides at first into two branches, one of these again giving off a third. These three branches leave the gland, giving off a number of small branches, which pass to their respective muscles, anastomosing with the sensory nerve of the face, forehead and neck.

The various branches as they are given off, also branches associating or communicating with other nerves, are as follows:

Interosseous Branches:

1. Great superficial petrosal nerve, which communicates with the fifth.
2. Anastomotic branch with tympanic plexus.
3. Stapedius nerve.
4. Chorda tympani.

After Leaving Foramen:

1. Posterior auricular nerve.
2. Digastricus nerve.
3. Stylohyoideus nerve.
4. Communicating with the glosso-pharyngeal.

Parotid Plexus:

1. Ramus coli.
2. Mandibular ramus.
3. Buccal ramus.
4. Zygomatic ramus.
5. Temporal ramus.

FUNCTION OF THE FACIAL NERVE.

The facial is the motor nerve of all the muscles of the face, including the platysma, but not including any of the muscles of mastication. It supplies the parotid gland, and through its communication by means of the petrosal with the vidian, some of the muscles of the soft palate, probably the levator palati

and azygos uvula (denied by some). By its tympanic branches it supplies the stapedius; through the otic ganglion the tensor tympani; through the chorda tympani it sends branches to the submaxillary glands, and to some muscle fibers of the tongue, through its connection with the lingualis. It supplies the mucous membrane of the anterior two-thirds of the tongue. By some of its branches before it comes upon the face, it supplies the muscles of the external ear, the posterior belly of the digastric, and the stylohyoideus. It, therefore, is a secretory nerve, as well as a nerve of special sense, aside from its motor functions. The sense of hearing is also affected by the muscles which it suplies, as the stapedius and tensor tympani. Again, the sense of smell is somewhat controlled by this nerve, in that it permits proper dilatation of the alae nasi; in this manner a proper amount of air is inspired. The sense of taste is supplied by the chorda tympani to the anterior two-thirds of the tongue. Some observers claim that this branch is an entirely independent nerve, only accompanying the facial, but having its origin from the nucleus of the fifth. Sight is influenced by its action on the lids.

The surgical anatomy will be described in connection with the operations.

CAUSES OF FACIAL PARALYSIS.

These will depend a great deal on the different portions of the nerve involved; for instance, the intracranial, the interosseous, or the external portion. However, one may have a paralysis due to the involvement of two of the portions, as an ascending or descending neuritis. Enumerating some of the most frequent causes we have, first, lesions, as:

1. Hemorrhages, subcortical, in the internal capsule, and before the communication with the nucleus.
2. Brain abscess.
3. Gumma.
4. Tumor.
5. Meningitis.
6. Suppurating disease of the middle ear, the most frequent cause, in the interosseous portion. It is most frequently in the region of the Fallopian canal, although it may occur anywhere along its course. Frequently it is associated with Bezold's mastoid abscess.
7. Traumatic, as following the mastoid operation (radical),

or labyrinthine exsection. It sometimes follows pressure from gauze packing, or improper manipulation of the protectors for the facial nerve. Fractures of the base of the skull are at times followed by facial paralysis, or gunshot wounds of the temporal bone.

In the external portion we find it associated with:

8. Tumors of the neck.

9. Retromaxillary tumors or abscesses.

10. Following resection of glands of the neck.

11. Osteoperiostitis of the lower jaw.

12. Tumors and abscesses in the parotid gland, and operations for these conditions.

13. Trauma, as stab wounds or the characteristic gashes of the German duelists, causes a goodly number of external facial paralyses.

The general systemic diseases that cause facial paralysis are:

14. Syphilis.

15. Rheumatism.

16. Other acute infectious diseases.

Among the largest group of cases are those spoken of as Bell's palsy, which are supposed to be due to:

17. Exposure to cold and draught.

18. Toxic.

According to others:

19. A severe gastro-intestinal toxemia may cause facial paralysis.

Such local causes as:

20. Very strong remedies poured into the ear, or applied, as chromic acid, trichloracetic acid, nitrate of silver, have been reported as causing paralysis.

21. Excessively hot or cold instillations into the ear with perforating drum membranes are given as causes of paralysis.

HISTOLOGY OF THE FACIAL NERVE.

A cross-section of the trunk of the nerve shows, as any other peripheral nerve, the following structures: (Fig. 2.)

Section (transverse) of the nerve trunk shows in the first place bundles of nerve fibres (funiculi) ensheathed by a fibrous capsule (epineurium). Each bundle is surrounded by its own sheath known as the perineurium. Each nerve fibre is surrounded by a sheath known as endoneurium. Between the structures are seen lymph spaces, veins, arteries, fat.

The termination of the nerve fibres is by the muscle plates, which is the axis-cylinders breaking up into a number of cells, which are practically fused with the sarcolemma of the muscle.

Section of Nerve Fibres (fig. 3):

(1) External (primitive nerve sheath) nucleated sheath of Schwann, or neurilemma.

(2) Intermediate or packing substance, also known as medullary or myeline sheath, or white substance of Schwann.

(3) Internally the axis-cylinder, otherwise known as primitive band, axis band, or axial fibre.

The neurilemma is structureless, and within it are seen at intervals nuclei, surrounded by protoplasm.

The medullary substance is supposed to be made up of two distinct parts: (a) a reticular framework, (b) fatty substance of a semi-fluid consistency, staining readily with osmic acid. The reticular network, according to some authors, has a highly chromatic property which is very resistant to chemical and digestive fluids, known as the neuro-keratin network.

The axis-cylinder consists of primitive fibrillae and can be traced from origin to termination.

Nodes of Ranvier. At regular intervals in the nucleated sheath of Schwann one can see a constriction. At these points the external sheath and axis-cylinder come in immediate contact. Between each two nodes of Ranvier is what is termed an internode. The latter varies in size, but averages about 1 mm. Only one nerve nucleus to each internode can be demonstrated. The constricting band of Ranvier is found between two internodes, uniting them to the external sheath, which band stains black with silver nitrate. Since the axis-cylinder also stains with silver nitrate, one can therefore at each node of Ranvier see a cross known as cross of Ranvier.

The size of a nerve fibre varies much in its course, the average being 6.2 to 8.7 micra.

PATHOLOGY OF THE FACIAL NERVE.

We may have either (a) neuritis or (b) death of the nerve. In the case of the former there is an increase in the vascular and plasmodic supply, with swelling of the component parts of the perineurium. There occur a round-cell infiltration and naturally a compression of the axis-cylinder. The degree of inflammation varies and in the marked cases may terminate

in permanent destruction of the nerve, that is, there may be gangrene or suppuration.

In the case of severance of the continuity of structure of the nerve, either by being cut or injured, as in fractures or gunshot wounds, in association with and complication of a suppurative process of the temporal bone, or tumors involving the nerve cell, one will see the following changes: According to F. Marchand,[1] the whole peripheral end of the nerve degenerates, as well as a small part of the central, up to the first node of Ranvier. On the second day after the division, the medullary sheath breaks down in the form of cylindrical fragments, which continue subdividing during the next few days. These fragments are still nucleated in the sheath of Schwann. The axis-cylinders also fragment, and are either swollen or fibrillary in aspect. They are included among the myelin fragments. At the same time a regenerative process begins in the nuclei of the sheath of Schwann; the nuclei enlarge, become richer in chromatin, and penetrate between the fragments of the myelin. Simultaneously, the protoplasm increases in volume and fills up the empty spaces between the drops of myelin. The latter are surrounded by protoplasm and form around nucleated lumps. The nuclei of the sheath multiply by karyokinesis on the third and fourth days, and subsequently become very abundant in numbers. The decayed medullary sheath and axis-cylinders, which are in the form of small, round drops, are often mistaken for fat. A great part of these nuclei, surrounded by protoplasm, transform these cells into long spindle elements, the importance of which is variously interpreted. This feature will be considered when the process of regeneration is taken up.

According to Ballance and Stewart,[2] the various steps in the process of degeneration are as follows: (1) Cellular change; (2) changes in the axis-cylinder and medullary sheath.

Cellular Changes.—The first sixteen or eighteen hours after the division of the nerve there is a diapedesis of leucocytes in the entire distal segment, and also in a small portion of the proximal segment. In from three to fourteen days, connective tissue cells replace these leucocytes. Similar to the invasion of the nerve with the leucocytes on the second day after the injury, there is a marked proliferation of the connective tissue cells, invading the entire distal segment and a small portion of the proximal. On the fourth day the fatty substance of the

myelin fragments is included within the substance of the connective tissue cells. The exact time in which the absorption is completed has not been determined, according to these authors. It is estimated, however, that the myelin disappears in about five weeks. After the complete absorption of the myelin, the connective tissue cells diminish and are outnumbered by the gradually increasing neurilemma cells. The remaining connective tisue cells become spindle-shaped and their destination is the formation of connective tissue fibers separating the chains of neurilemma cells. On the second day after the injury the neurilemma cells proliferate. At first their proliferating activity is greater than that of the connective tissue cells, and during this period they exert an absorptive action upon the degenerating medullary sheath. Later, their proliferating activity is surpassed by the connective tissue cells, the former continuing to proliferate in columns. The young neurilemma cells preserve the original longitudinal direction of their parent cells, and from their opposite poles they shoot out fine protoplasmic processes which gradually increase.

Changes in the Medullary Sheath.—Decay of the axis-cylinders begins on the fourth day, and is very pronounced on the seventh. Fine fibers have a greater resistance to disintegration than the coarse ones. The fragments of the axis-cylinders are absorbed by the connective tissue cells principally, and partially by the neurilemma cells. The degenerative process is not influenced by immediate suture, as it takes place uniformly. The muscle supplied by the divided nerve rapidly diminishes in weight.

Regeneration.—This begins, according to Ballance and Stewart, before degeneration is completed. At the end of the third week the peripheral segment contains a great number of elongated spindle cells, which shoot out axis-cylinders from their poles. The scar tissue contains cells whose ends interlace in an irregular manner, and form a new network of new axis-cylinders. The anastomosis of the axis-cylinder of the two ends of the nerve is accomplished at the end of four weeks.

SYMPTOMS.

These will depend on the location of the lesion, with reference to the involvement of other nerves or parts of the body. There is, however, a characteristic picture of a facial paralysis that can not be mistaken very well for any other condition,

although we may have different degrees of paralysis, as a mild paresis or complete paralysis.

The characteristic picture (Fig. 4) shows the angle of the mouth drooped, the usual nasolabial fold obliterated; in fact, the whole cheek hangs flabby. The lower eyelid sags and the upper one lies closer to the eyeball; the eyebrow seems lowered as well as the skin covering the forehead—the usual creases of this region are wanting. Tears are found gathered in the corners of the eyes, and often seen trickling over the lower lid. The bulbar conjunctiva is frequently injected. So far as the patient is concerned, there is a most unpleasant appearance and sound when he begins to speak, to laugh or attempts to move the facial muscles. He can not close the eye, and when forced to do so, the eyeball rolls up, and one sees only the scleral portion. Asked to whistle or to blow up the cheeks, one finds that it is impossible for him to do so. The air will escape through the paralyzed side of the mouth. Patients complain a great deal of their food lodging within their cheeks, and find considerable difficulty in clearing the buccal surfaces. They generally use the other side of the mouth for mastication. Inasmuch as most of the cases are those where the nerve is injured above the chorda tympani, there are symptoms of perverted or loss of taste. A complaint frequently made is that of a metallic taste. The eye symptoms bother the patient a great deal, especially when exposed to the wind, and on first arising in the morning.

PROGNOSIS.

This depends absolutely upon the cause and location of the lesion. Suffice it to say, however, that in general the prognosis is bad and the course is a long and protracted one, except in that group of cases known as Bell's palsy, such as are classed as toxic neuritis, rheumatic, luetic or compressive. In the latter cases, when the condition is relieved, recovery takes place more readily, and even here one must give a very guarded prognosis. I have had a number of cases of the above type that resisted all sorts of local and general treatment, and only after three years did complete recovery take place. However, most of them recovered much sooner, between three weeks and six months.

Again, the prognosis depends on the treatment. I mean, specifically, surgical treatment, and when instituted. To be more specific, if one has a complete paralysis, with a positive reaction of degeneration, it is best not to wait too long before

operating for fear of losing the muscular action, as the muscles tend rapidly to undergo trophic changes and waste away, after which an operation on the nerve will be practically useless. Another important point is the onset of paralysis, especially after operation. If it be gradual and take two or more days before development, the progress is better than if it has occurred as soon as patient comes out of the anesthetic, which condition is more grave.

REACTION OF DEGENERATION.

Normally, the facial, like other motor nerves, is affected by electrical stimulation as follows: The faradic current, when applied over the trunk of the facial, behind the ear or over the point of emergence of its branches on the face, causes a quick contraction of muscles supplied by these nerves. The electrodes may be applied directly over individual muscles with the same result. When a sufficient amount of galvanic current is applied over the nerve or muscles, contraction occurs every time contact is made, with the so-called "make" of the current; slightly stronger currents produce another contraction when the current is released or "broken." In all these stimulations the "cathode" or negative pole has been found to be the more active; that is, less current is required to get a contraction when the negative pole is placed upon the muscle to be stimulated; while the positive electrode may be put upon an indifferent point, preferably the sternum or back of the neck. The normal formula usually given is:

C. C. C. > A. C. C. = A. O. C. = C. O. C.

In which C. stands for cathode, A. for anode, C. for closing, C. for contraction, O. for opening.

C. C. C., cathode closing contraction.

> A. C. C., greater than anode closing contraction.

What do we mean by the reaction of degeneration, which is abbreviated by R. D.?

In this, the important points are that faradism fails to produce a contraction both through muscles and nerves, while galvanic currents produce no contraction of the muscles when applied over the nerve, but still cause muscular contraction when applied directly over the individual muscle. There is, however, a change in the character of the contraction. It has ceased to be prompt, lightning-like, but has become sluggish, peristaltic-like. A minor point is the inversion of the polar reactions. Whereas in the normal muscle the cathode is the

more active in closing or making of the current, in the degenerated muscle the anode may be more active in closing and less than normal in the opening contraction, that is, the reverse of the normal muscle. This change, however, is unimportant. The time of occurrence of the reaction of degeneration is from eight to ten days following injury or disease of the facial nerve.

Immediately following injury, the nerve response may be even greater than normal; that is, less current is required to produce the effect. Authors have differentiated various degrees of reaction of degeneration according to the completeness or incompleteness of the reaction formula. The prognosis, therefore, is somewhat governed by the fact as to whether reaction of degeneration is complete or incomplete. The less complete the R.D., the better the prognosis.

TREATMENT.

The treatment must necessarily again depend on the etiology and pathology, as well as on the location of the lesion. In a general way, however, we can say the treatment is either expectant, local, general, medicinal, mechanical, or surgical. Taking up the cases systematically, as mentioned in the etiology, I will state that the local treatment will constitute in the first place, removing, if possible, whatever may be pressing on the nerve, as tumors, abscesses, necrotic bones, sequestra, and other foreign bodies. One of the frequent procedures necessary is a mastoid operation, and the removal of cholesteatomatous masses, sequestra and granulations over the region of the facial canal. Quite a number of cases of this sort will get well after such procedure.

Facial massage, vibratory massage, etc., may be systematically carried out, twice or three times daily for a half an hour at a sitting.

Electricity—a mild faradic current, is to be used as follows: The positive pole is applied over the sternum, and the negative used as the stroking electrode over the whole paralyzed area, once daily for three to five minutes, followed by galvanic current, the technic practically the same, using from five to ten milliamperes for three to five minutes.

The eye must be protected from external influences, as foreign bodies, dust and wind will irritate the condition; usually I give the patient a shield or a large lens (plain). Over night the eye should be tied up. A drop of argyrol, 5 per cent, and

boracic acid irrigations are good adjuvants to prevent infection.

Thorough cleansing of the mouth and especially the inner surface of the cheek of the paralyzed side, as food particles remaining therein produce considerable irritation from decomposition.

The general treatment consists in tonics, particularly strychnin sulphate, 1/60 of a grain three times a day.

Eliminating and reabsorptive remedies should be used, as cathartics, sweat baths, potassium iodid solution.

A very wholesome nutritious diet, such as will leave the least residue, is to be advocated; rich condiments and red meats are to be restricted.

Strict attention to hygiene and exrcise is to be encouraged, particularly in the open air. A change of scene is to be recommended, that is, away from friends and acquaintances, because patients are very frequently sensitive about these conditions, and very often become morose, this condition causing delay in the recovery.

Of course, if it be a case of leutic facial paralysis, a thorough anti-syphilitic treatment by mercury and iodid of potash is to be instituted, associated with all the other general and local treatments required in that condition.

SURGICAL TREATMENT.

The indications for an operation on the facial nerve are confined to absolute paralysis, no matter from what cause and wherever the location of the lesion (lower segment), after all other methods have failed. The procedures are usually anastomosis with other neighboring motor nerves, as hypoglossal, spinal accessory and glossopharyngeal, in the order named.

The contraindications are: (1) When the general condition of the patient will not warrant such an extensive operation, and (2) when the other nerves, as hypoglossal, spinal accessory and glossopharyngeal, are not in good condition or accessible. (3) In cases of congenital absence of the facial muscles or nerve. (4) In long standing facial paralysis where one, in making the test of reaction of degeneration, can safely say that all the muscles supplied by the facial nerve are wasted and beyond the possible chance of regeneration. This last contraindication must, however, be modified in as much as

there are cases on record (Hackenbruch) in which the above conditions existed, which were operated upon and recovered.

The historical data of neuroplasty may be briefly stated as follows: Probably Ballance and Stewart were the first to have united the facial and spinal accessory nerves for the cure of facial paralysis, in 1895, but did not publish it until 1903.[4] In 1898, at the suggestion of Fouret, Foure[5] united the facial with the trapezius branch of the spinal accessory. Experimentally, P. Manasse,[6] in 1900, and about the same time Barrago Ciarello[7] accomplished satisfactory results on animals in uniting the facial with the spinal accessory. Robert Kennedy,[7] in 1900, operated on a man by uniting the facial with the spinal accessory. Harvey Cushing[8] performed the same operation in 1902. After that, quite a number of similar operations followed. The hypoglossal, however, was not used until about 1902-3, as cases reported by Ballance and Stewart (*British Med. Journal*, May, 1903). Koerte,[9] Nicoll,[10] Frazer,[11] Bardenhauer,[12] Schaefer,[13] of Edinburgh, suggested the union of facial and glossopharyngeal.

Herewith follow the cases tabulated by Murphy, with some additions by myself from literature and personal communication as well as cases operated on by myself:

NERVE ANASTOMOSIS FOR THE CURE OF FACIAL PARALYSIS, TIC, OR SPASM.

A.—Experimental.

CASE 1.—Paul Manasse. (Munk's Laboratory.) (*Arch. f. klin. Chirurgie*, vol. lxii, p. 822.) Seven dogs operated on, but only five considered in final results. Spinal accessory freed from its implantation into sternomastoid muscle to atlas; end-to-end anastomosis of central spinal accessory with peripheral facial divided at the stylomastoid foramen. Four silk sutures passed through quarter of the thickness of spinal accessory.

Results.—Clinical results not sufficiently satisfactory. In first three months there was no faradic response; the latter obtainable after four months. Electric stimulation of central spinal accessory was followed by response in peripheral facial. Histologically, new axones penetrated the facial from the central spinal accessory. Manasse thinks that "months" is an in-

sufficient time for voluntary motion and disappearance of atrophy. "Years" would lead to perfect restoration.

CASE 2.—Barrago Ciarello. (*Policlinico,* 1901, No. 3.) In two dogs. (a) Anastomosis between central end of spinal accessory and peripheral of facial. (b) Anastomosis between the central end of vagus and peripheral end of facial.

Results.—(a) Restoration of function commenced after four months, and was almost completely restored in six. There was still some muscular atrophy left. Reaction of degeneration disappeared. (b) Same results. Ciarello believes that spino-facial and vagofacial anastomosis is promising in man in recent facial paralysis.

B.—Clinical.

CASE 1.—Charles A. Ballance, Hamilton Ballance, and Purves Stewart. (*Brit. Med. Jour.,* May 2, 1903.) Facial paralysis. Following operation for suppurating mastoiditis. Spinofacial anastomosis. (Operation by C. A. Ballance.)

Results.—After several years, muscles responded to galvanic and faradic currents. Still some drooping of angle of mouth and widening of palpebral aperture. Movements always associated; voluntary independent movements of muscles of face not possible.

CASE 2.—Ballance and Stewart. Facial paralysis. Otorrhea of twenty years' standing. Spinofacial anastomosis performed. (Operation by C. A. Ballance.)

Result.—Some improvement after ten months.

CASE 3.—Ballance and Stewart. Facial paralysis. Following operation for otitis media. Spinofacial anastomosis performed (after six months). Anastomosis of central end of spinal accessory with side of facial. (Operation by H. Ballance.)

Results.—All muscles of face responded to faradic currents after four weeks. Three months later, some voluntary motion. Three sutures of French silk used.

CASE 4.—Ballance and Stewart. Facial paralysis. Following operation for otitis media. Spinofacial anastomosis, four months after the initial paralysis. (Operation by C. A. Ballance.)

Results.—Faradic response after nine months. Associated movements only. After twenty-one months, voluntary independent elevation of inner end of eyebrow.

CASE 5.—Ballance and Stewart. Facial paralysis. Fracture of skull, due to a fall of fifteen feet. After thirty-five months. Spinofacial anastomosis. (Operation by C. A. Ballance.)

Results.—Slight voluntary movements in muscles of face if shoulder was elevated under resistance.

CASE 6.—Ballance and Stewart. Facial paralysis. Following operation for otitis media. Spinofacial anastomosis six months after initial paralysis. (Operation by C. A. Ballance.)

Results.—No improvement; no electric response; no return of independent voluntary or associated movements.

CASE 7.—Ballance and Stewart. Facial paralysis. Following the removal of a cystic tumor of lower jaw. Operation twelve months after initial paralysis. (By C. A. Ballance.) Anastomosis of peripheral end of facial with side of hypoglossal.

Results.—No improvement at the time of operation. Atrophy of half of the tongue.

CASE 8.—Faure. January 28, 1898. (a) *Gazette des Hopitaux,* 71eme annee, 1898, p. 259. (b) *Revue de Chir.,* 1898, t. xviii, p. 1098. Facial paralysis. Injury. Eighteen months later, anastomosis of facial with accessories.

Results.—Slight improvement some time after the operation. "Half successful."

CASE 9.—Robert Kennedy. Phil. Trans. of the Royal Society, series B, vol. cxciv, p. 127. Facial tic (facial hemispasm). Spinofacial anastomosis. End to side.

Results.—Cured. After 470 days, patient able to close eye. Muscles regained volume, and contracted voluntarily.

CASE 10.—Hackenbruch (Wiesbaden). *Verhandl. der deutschen Gesellschaft für Chir.,* 1903, bd. xxxii, p. 231. Facial paralysis. Sudden paralysis of facial, three months' standing. Seven and three-fourths years after appearance of paralysis, Spinofacial anastomosis.

Results.—Improvement after four and one-half months. After nine months, patient could draw the angle of the mouth downward.

CASE 11.—Körte. (*Deutsche med. Woch.,* April 23, 1903.) Facial paralysis. Following operation for ostitis of mastoid process. Hypoglosso-facial anastomosis.

Results.—Good after one and one-half years, but associated movements present.

CASE 12.—W. W. Keen. Phila. Neurol. Soc., Feb. 24, 1903. (*Jour. of Nerv. and Ment. Dis.*, Feb. 24, 1903, p. 366.) Facial paralysis. Struck by a glass bottle. Five weeks after accident, Spinofacial anastomosis.

Results.—"Perfectly smooth recovery."

CASE 13.—Harvey Cushing. (*Ann. of Surg.*, May, 1903.) Facial paralysis. Revolver-shot behind ear, involving mastoid. Operation forty-three days later. End-to-end spinofacial anastomosis.

Results.—Complete restoration of function.

CASE 14.—Hackenbruch. (*Arch. f. klin. Chir.*, 1903, Bd. lxxi, p. 631.) Facial paralysis of seven years' and nine months' standing. Spinofacial anastomosis by implanting two-thirds of spinal accessory into facial.

Results.—Electrical response in paralyzed muscles. Voluntary contraction independent of those of trapezius.

CASE 15.—James H. Nicoll. (*Lancet*, 1903, p. 956.) Convulsive tic. Anastomosis of facial with hypoglossal.

Results.—No improvement at the time of communication.

CASE 16.—Szeyner. (Abstracted in *Fortschritt der Mediz.*, 1903, p. 1060.) Facial paralysis. Shot; attempt at suicide. Facial anastomosis with a flap taken from spinal accessory.

Results.—Voluntary contractions after three months; able to close eye, and whistle.

CASE 17.—Charles H. Frazier. (*Univ. of Pa. Med. Bull.*, 1903-4, vol. xvi, p. 306.) Facial paralysis. Gunshot wound. End-to-side anastomosis between peripheral facial and side of hypoglossal.

Results.—Too recent to report conclusively on the case.

CASE 18.—Glück. (*Berl. Gesell. f. Psychiatrie*, May 11, 1903.) Facial paralysis. Division of facial during mastoid operation for otitis media. End-to-side anastomosis between peripheral facial and side of spinal accessory.

Results.—Regeneration of facial. Associated movements.

CASE 19.—A. I. Bouffleur, Chicago. Personal communication. Facial paralysis due to gunshot wound (attempt at suicide), June 14, 1904. The tip of mastoid was cut off by the bullet. Division of lower branches as well as of common trunk of facial. End-to-end anastomosis between spinal accessory and facial, performed four weeks after injury.

Result.—Case lost sight of.

CASE 20.—Dr. O. Facial paralysis. Following operation

for otitis media. March 10, 1904. End-to-end spinofacial anastomosis.

Results.—Muscular atrophy not improved. Patient has associated movements only in muscles of face.

Same case as above. J. B. Murphy. Facial paralysis. Following operation for otitis media. March 10, 1906. Freed nerve from scar tissue and imbedded it in muscle.

Results.—Muscular atrophy improved, and voluntary motion of face is manifest. The associated movements continue. Patient was reoperated on by the author.[1]

CASE 21.—Löhlein. (Communication; *Vereinigung der Chirurgen,* Berlin, July, 1904.) Facial paralysis. Otitis media. Anastomosis of facial with spinal accessory.

Results.—"Excellent results." No associated movements.

CASE 22.—Bardenheuer. (*Münch. med. Wochenschr.,* 1904, No. 28.) Facial paralysis of ten years' standing. End-to-end anastomosis between peripheral facial and side of hypoglossal.

Results.—"Very good." Tongue paralyzed. No associated movements.

CASE 23.—Bardenheuer and Lambeth. (*Festschrift zur Eröffnung der Akademie für praktische Medizin zu Köln,* 1904, p. 219. Facial paralysis. End-to-end anastomosis between peripheral facial and side of hypoglossal.

Results.—Good.

CASE 24.—Mintz. (*Zentralbl. f. Chir.,* 1904, No. 22, p. 684). Facial paralysis. Trauma during mastoid operation for otitis media. Anastomosis of facial with spinal accessory.

Results.—Complete restoration of motor function. Good closure of eye.

CASE 25.—Villar. (*Gaz. hebd. des. Sc. Méd.,* Bordeaux, Feb. 7, 1904.) Facial paralysis. Trauma to facial during mastoid operation for otitis media. Anastomosis of facial with spinal accessory.

Results.—"The results were astonishing, as patient was able to move his lid on next day after operation. This rapid improvement, however, did not remain permanent."

1. **The point of anastomosis found surrounded by great mass of connective tissue.**

Electrical irritation of central end of spinal accessory produced contractions in muscles of the face, showing that new fibers had grown from the spinal accessory into facial, but that impulses could not travel, on account of surrounding connective tissue. Point of anastomosis was freed and protected by soft tissues. Good results.

CASE 26.—Alexander. (*Wien. med. Wochenschrift*, No. 11, 1904.) Facial paralysis. Otitis media paralysis of five years' standing. End-to-side anastomosis between divided facial and side of hypoglossal.

Results.—Paralysis of half tongue. No improvement at time of communication.

CASE 27.—Alfred S. Taylor and L. Pierce Clark. (*Jour. Am. Med. Assoc.*, March 24, 1906.) Facial paralysis. Suppurative mastoiditis. The peripheral segment of facial inserted into a slit of hypoglossal.

Results.—Improvement after twenty-five months. The anastomosis was protected by Cargile membrane.

CASE 28.—Same. Facial paralysis. Paralysis followed a severe earache. Same operation.

Result.—Improvement.

CASE 29.—Same. Facial paralysis. Suppurative mastoiditis. Same operation.

Result.—Improvement.

CASE 30.—Same. Facial paralysis. Bell's palsy. (Paralysis twelve years' standing.) Same operation.

Results.—Improvement. Although paralysis existed for twelve years, facial nerve was normal in size and aspect.

CASE 31.—Same. Facial paralysis. Suppurative mastoiditis. Same operation.

Result.—No improvement.

CASE 32.—A. I. Bouffleur, Chicago. Personal communication. Facial paralysis resulting from fracture of mastoid, concomitant with fracture of skull (1903). Nerve-trunk intact, as far as continuity is concerned. End-to-end anastomosis between spinal accessory and facial. Performed September 11, 1906.

Results.—Some improvement shortly after operation, inasmuch as fluids did not run out of the mouth from paralyzed side. Time too early to give definite results.

CASE 33.—J. B. Murphy. Present paper. Facial paralysis. Division of facial during mastoid operation for otitis media. End-to-end anastomosis between peripheral facial and central spinal accessory.

Results.—Evidence of returning motion pronounced. See history. Too recent for complete recovery.

Spiller, University of Penn., *Med. Record*, November, 1903.

Rothmann, *Berliner klin. Woch.*, 1903, No. 34.

Bernhardt, *Berliner klin. Woch.*, 1903, No. 34.

Koster, *Berliner klin. Woch.*, 1903, No. 34.

Gradenigo, *Laryngoscope*, 1907.

McArthur, Eisendrath, Ochsner, Chicago surgeons, have done this operation, but for some reason have not reported their results.

Beck, J. C., five cases of facial hypoglossal anastomosis. (Present paper.)

GENERAL POINTS AS TO NERVE ANASTOMOSIS.

In the first place, a clean, aseptic field for primary union of the sutured ends is practically necessary to obtain rapid regeneration through the anastomosed ends; proper protection (as shown in Murphy's cases) of the anastomosed ends, to prevent access of cicatricial formation about them, by placing into a neighboring muscle; absolute rest to the parts, almost immobilization of the head for the first few days after operation, to prevent severance or traction on the united ends.

The methods of anastomosis most frequently employed are:

1. End to end.
2. End to side.

The most preferable mode is that of implanting the exposed stump on the paralyzed side into a niche of the living nerve; this niche should have its axones about 1/2 to 2/3 cut transversely, in order to obtain direct contact and union, and, secondly, so that absolute loss of function does not result in the muscles supplied by the live nerve, as, for instance, the tongue would only be partially paralyzed when the hypoglossal is used.

SUTURE OF NERVES.

Either primary or secondary. By "primary suture" is understood suture of the two divided ends immediately after the accident or within the first twenty-four hours. Secondary suture is the approximation of the two divided ends within several months or years after the injury. The older surgeons base their practice on the theory that a peripheral segment which has been disconnected from the central for a long period of time is permanently degenerated, and an attempt at union would be futile. The most modern surgeons, better informed on the histology and physiology of the peripheral nerves, and especially those who believe in peripheral regeneration, are of the

opinion that new nerve fibers regenerate in the peripheral segment, even if it has remained disconnected for a considerable period of time. The fibers are imperfect, and as soon as they are united with the central end, under the influence of central impulses they develop to anatomic perfection and to normal physiologic function. The limitations of time in which this restoration may take place has not been definitely determined, but restoration of functon has been secured many years after division.

In any operation upon the nerve trunks, one must, first of all, identify the nerve; otherwise the operator is in the dark, and the final result will undoubtedly be compromised. This identification can easily be accomplished by making use of the nerve excitor. This is composed of a metallic cylinder which can be attached at one end with the poles of a faradic battery, while the other end is provided with two platinum needles or wires, three centimeters in length, and situated a distance of half a centimeter from each other. These tips can be easily sterilized, and should always be at hand when operating upon the nerve trunks. The importance and the value of this instrument can be appreciated when we think of the conditions of the tissues in an old and extensive injury in which they are all matted and bound together by connective tissue. The structures have lost their natural color, and the anatomic relations and identification of nerve trunks becomes an impossibility without the use of the excitor.

Operative Procedure.—The first essential is the removal of all connective tissue that caps the nerve endings to a degree sufficient to expose clearly the fasciculi and neurones, even though this shortens the nerve beyond the possibility of direct end to end suture. The axis-cylinders or nerve fibers will span a considerable distance under favorable conditions, but they will not penetrate connective tissue masses, so that it is absolutely necessary to have all the cicatricial or new-formed connective tissue removed from the nerve ends. The failure of this removal has contributed more than any other element to the failure of restoration of function after nerve suture. This, of course, has reference to secondary suture.

The needle used for suturing should be a round intestinal needle, without cutting edges. Suture material should be fine linen, silk, kangaroo tendon, or slowly absorbable catgut. The suture should not be passed through the substance of the nerve,

but through the perineurium, and also through the surrounding connective tissue. This point is very essential for success. The trauma produced by passing the needle through the substance of the nerve, and especially if a slight infection takes place, causes a proliferation of connectice tissue which compromises the restoration of function. Figure 5 shows the various methods of suture.

If the nerves face each other, and are not separated by a considerable gap, a slight traction established when the muscles are completely relaxed, will make it possible easily to approximate the ends. A little stretching of the ends can be easily made without any prejudice to the ultimate result. If the distance is considerable between the ewo ends, the nerves can be united in several ways. Létievant is the originator of "suture a lambeaux," or flap-suture. Foreign bodies, nerve segments from other animals, portions of segments of spinal cord from small animals, have been interposed between distant segments. In the last ten years, in cases of considerable distance between ends, the divided peripheral end was implanted into a neighboring healthy nerve, which was only partially divided. In several instances, especially where the trauma has compromised the nerve and bone, in order to be able to approximate the two ends a segment of bone was resected, that the divided ends might be brought in contact (Keen, Trendelenburg). Büngner interposed a piece of human brachial artery between the segments of the peripheral nerve in a dog. After 43 days the space between the two divided ends of the sciatic was filled with new nerve fibers. Bungner ascribed to the artery the role of a director and medium along which the nerve fibers can travel in a straight direction. Nothaft interposed a segment of rabbit's aorta between the two ends of the sciatic, from which he had excised one-half centimeter. When the gap between two ends is considerable, the contact can be established by foreign material. In a case of injury to the radial, Glück, in 1888, interposed catgut bundles between the ends, which were separated by a gap of 5 cm. The gap was filled in six months, or, as he says, there was a neurotization of the catgut bundles. The result was good.

On the strength of Forsman's observation on "positive neurotropism," several surgeons and experimenters have inserted the two ends into a tube of decalcified bone, previously sterilized; this has enabled a more rapid union of nerve fibers.

"Suture a Distance."—This procedure originated with Assaky (Roumania). By this is meant the interposition of catgut fibers between the two ends of a divided nerve where a considerable gap exists. In Assaky's experiments (4 dogs, 2 rabbits), in the dogs favorable results were obtained in from 67 days to 4 months. The greatest segment successfully excised was 3 cm. Once he resected 6 cm. and interposed catgut fibers between the ends; no regeneration occurred after 39 days. If the distance is considerable between the segments, a piece of muscle may be interposed, or the two segments may be imbedded in a muscle from the vicinity in such a manner as to place the cut surface of each end face to face. This has been done by Levings, of Milwaukee, and several times by Murphy. The ideal results in nerve suture are obtained, as in the repair of all other tissues, by an exact juxtaposition of similar histologic elements, end to end. This requires the least interposition of new-formed connective tissue, and in nerve union it is of special importance.

Protection for United Nerves.—The ends of the nerves which are either in perfect apposition or face each other from a distance may be protected from the invasion of connective tissue from the side by sterilized tubes. Decalcified bones were used by Vanlair. Payr used tubes of magnesium. Lotheisen prefers to protect the nerves with tubes of gelatin previously hardened in a two per cent solution of formalin. The gelatin tubes, after being treated by this solution, are preserved in 96 per cent alcohol; the lumen should be from 4 to 9 mm. Gelatin tubes can be sterilized for two hours by dry heat of from 110° to 120°. It has been my practice to protect the nerves from invasion of connective tissue by covering the point of suture with fascia, muscle substance, or fatty tissue. In the wrist or ankle areas, where muscle and fat are not available, gelatin and decalcified bone are good materials. The blood vessels of animals, always at hand, are good for this purpose. A mass made of equal parts of oil of sesame and paraffin, with a melting point of 107° is especially good. This can be flattened out with a spatula almost as thin as paper and wrapped around the point of union, completely sealing the ends so that the neuroglia can supply the only immediate agglutinative material, which is readily penetrated by the axones.

Foramitti protected the points of anastomosis by surrounding them with arteries of the calf. The union is always perfect,

and the point of anastomosis entirely free from the connective tissue in the vicinity. There is an insignificant adhesion between the intima of the artery and the sheath of the nerve. The arteries are prepared for this purpose in the following manner: A segment of artery from a calf is placed on a glass rod and hardened in 5 to 10 per cent of formalin solution for 48 hours, then it is washed in water for 24 hours, afterwards boiled for 20 minutes in water, and finally preserved in 95 per cent alcohol.

An ever-available material for this purpose is the membrane lining the inner surface of an egg-shell. The Cargile membrane may be used for the same purpose, as may also the peritoneum of a rabbit. Recently Murphy conceived the idea of wrapping around the point of anastomosis a roll of paraffin tape or a mixture of paraffin and wax, or simply a wax tape; however, these foreign materials were never used when a muscle or fascia could be obtained, either by flapping or splitting the muscle and inserting it where a normal muscle belly is directly available. The use of inorganic material is always somewhat hazardous.

THE SURGICAL ANATOMY OF THE FIELD OF OPERATION.

(Fig. 6.)

1. *Facial-Hypoglossal Operation.*—After the skin and superficial fascia and platysma are retracted, a number of small veins are encountered, branches of the anterior and posterior facial, external and internal jugular. The anterior border of the mastoid process is located, and the sterno-mastoid muscle. Then the parotid gland is exposed with the ramus and angle of the lower jaw. On separating the sterno-cleido-mastoid muscle backward, one comes to the posterior belly of the digastric, which, when drawn under the lower jaw, will expose the submaxillary gland. Now the common sheath of the internal jugular vein is seen with the internal and external carotid arteries below and anterior to it. On slight retraction backwards of the sterno-cleido-mastoid muscle and internal jugular, one can see shining through a loose areolar tissue, a whitish band, about one-eighth of an inch in diameter, which, when laid bare, is the hypoglossal nerve. This can be followed under the posterior belly of the digastric. At this point it lies on the internal carotid and some of the branches of the external carotid,

as it comes down and curves forward, particularly the occipital artery.

The parotid region will show on its posterior border, the facial nerve entering and passing transversely under the auditory meatus to the anterior border, and under surface of the mastoid process. It lies at this point on top of the stylo-hyoid muscle, consequently the styloid process, which can always be felt. The transverse process of the atlas is situated at this point, and is the landmark or location of the beginning spinal accessory nerve.

2. *Facial-Spinal Accessory Operation.*—At first, the same structures come to view, but in order to obtain the view of the spinal accessory the internal jugular is drawn forward or towards the larynx, and the sterno-mastoid muscle is raised, when the upper branch, or the one supplying the latter muscle, is seen, and the greater branch passes down to the trapezius. (Shoulder.)

TECHNIC OF THE FACIAL-HYPOGLOSSAL ANASTOMOSIS.

After the usual local as well as general preparation the patient is placed under a general anesthetic.

Instruments.—Two knives, one regular size and one very small; one dozen fine artery forceps; two pairs of tissue forceps; two pairs of anatomic forceps; one Kocher director; one simple grooved director; two small dull retractors; two larger dull retractors; one aneurysm hook; two pairs scissors, one straight, one curved; one pair of fine-pointed conjunctival forceps; one special facial nerve dissecting instrument (Fig. 7); one-half dozen fine small needles (round); one needle-holder.

Material.—Sutures, finest of silk, catgut, silkworm gut, decalcified chicken bone tubes, Cargile membrane, paraffin tape.

An incision is made, beginning close back of the auricle, midway between its upper and lower margins, carried downwards and slightly forwards in the direction of the hyoid bone. The length of the incision should be about four inches, and carried at first through the skin, superficial fascia and platysma muscle. These cut margins should be dissected loose and retracted. The external jugular vein, with its anterior and posterior facial veins, is exposed, ligated above and below, and severed. The deep fascia is carefully incised, preserving any of the branches of the superficialis coli nerves; the anterior

border of the sterno-cleido muscle is exposed and followed upwards to the tip of the mastoid process. The posterior belly of the digastric is next exposed and the posterior border of the parotid gland by forcibly retracting the auricle and the skin over the ramus of the lower jaw forward. The anterior border of the mastoid process is followed along towards the tip into the depth, so as to reach the deeper layer of the parotid gland and to look for the entrance of the facial nerve into the latter, about on the level of the lower border of the external auditory meatus. The greatest mistake is made in looking for the facial nerve too near the surface and too far down from the ear. (One often finds here a superficial cervical branch.) After finding the facial nerve, which is recognized as a whitish cord, about the size of a Eustachian catheter No. 2, it is hooked up by means of the aneurysmal hook, and followed backward and upwards towards the stylo-mastoid foramen. This can best be located by following back the posterior belly of the digastric and the stylo-hyoid muscle immediately under it. The highest point of these two muscles will locate the foramen and the exit of the facial nerve. (Fig. 8.) In order to follow the nerve as high up into the foramen as possible, I have adopted and modified the ring instrument as devised by Mayo for dissecting varicose veins subcutaneously, and have added to it its ability to grasp. It is used in the following manner: Slip the ring over the nerve and follow it down to the foramen as high as possible and pull it out of canal. Place a sponge soaked in warm salt solution about it and allow it to remain in the field. Next the hypoglossal is located by retracting the posterior belly of the digastric upwards under the lower jaw and the sterno-cleido-mastoid backwards. The common sheath of the vessels is exposed. This, with the sterno-cleido-mastoid muscle, is bluntly retracted. By bluntly dissecting some of the loose areolar tissue over the internal and external carotids, which can be felt pulsating with the finger, one can see a whitish structure passing over the external carotid just where the occipital artery is given off. This is the hypoglossal nerve which can be traced at this turn under the internal jugular and internal carotid upwards for a short distance, where it is lost sight of in its direction towards its foramen vertically into the cranial cavity. The distal course of the hypoglossal is forwards towards the tongue. At this point the technic will differ, whether we do an end to end or end to side anastomosis.

End to End.—(Fig. 9.) Follow the hypoglossal in its forward course to the tongue. The same ring-formed scissors may be used as for the facial. The nerve should be severed at the point, so as easily to meet the facial stump. The next step is to bring the cut ends into perfect apposition and suture them. One can use any of the methods described in the suture of nerves. The anastomosed nerves are now carefully placed into a pocket made within one of the muscles, preferably the posterior belly of the digastric, and a few superficial catgut sutures inserted, closing this pocket. All the structures of the neck are carefully replaced in their anatomic positions and wound closed without drainage, unless pus has been encountered, as in a suppurating gland, etc.

End to Side.—(Fig. 10.) Bring down the amputated facial nerve stump and find the location on the hypoglossal where the anastomosis will be best, that is, without producing any tension on either the facial or hypoglossal nerves. Having found that point, an incision, one-quarter inch long, is made with the small knife into the nerve sheath parallel with the course of the nerve. With the same knife about one-half of the axis-cylinders are several within the sheath, that is, transversely, in order that when the facial stump is later implanted there will be direct axone contact, and thereby proper union, and still some of the fibers be left intact to perform part of the hypoglossal function. The next procedure is the preparation of the facial stump. The nerve sheath should be slit for about one-eighth of an inch and retracted so as to expose the stump, that is, its axis-cylinders. The sutures are placed within it, but only through the sheath. Any method may be selected for suture of the nerves utilizing the slit. Fine conjunctival forceps are used by the assistant to separate this slit by allowing them to spring open. After the sutures are tied, these anastomosed nerves (fig. 11) are placed within a pocket in a muscle (posterior belly of digastric) and treated in the manner already indicated. The wound is closed in the same way as in the other procedures.

TECHNIC OF THE FACIAL-SPINAL ACCESSORY ANASTOMOSIS.

The first steps of the preparation and operation are the same, except that the skin incision is not made so far curved forward, because the spinal accessory nerve lies more posterior than the hypoglossal. Locate the transverse process of the atlas; by

well retracting the sterno-cleido-mastoid at the mastoid tip, one can hook up the spinal accessory nerve. If it is decided to use the trapezius branch, then that is dissected and severed, brought upwards and united to the facial. The anastomosed nerves are then placed within a pocket formed within the sterno-cleido-mastoid, or the posterior belly of the digastric muscle, and secured with two catgut sutures to prevent cicatricial formation at the new-formed union of the nerves, thereby preventing bad results.

POST-OPERATIVE OBSERVATIONS AND TREATMENT.

There are considerable soreness and pain in the regions operated on, and patients complain of some difficulty in swallowing and speaking for the first day or two. This is especially so if the hypoglossal is chosen. If closely questioned, most of the patients will state positively that they feel a sort of a tone in the paralyzed side of the face, but this soon disappears, and does not return until later, when gradual regeneration has taken place. Patients should be kept absolutely at rest for about a week, and as little motion of the head as possible should be made in order to insure good and rapid union of the nerve ends. The diet may be regular after the first forty-eight hours, if the patient is able to swallow well. The stitches may be removed at the end of a week or ten days.

Electric treatment should be begun as soon as the patient is walking about, in order to keep up the strength of the muscles and to stimulate the nerves. Mild faradic and galvanic currents are used as before operation. In case the hypoglossal is used, about the first signs that one observes of return of function are a sort of contraction of the angle of the mouth, especially during the act of swallowing, and this may be observed as early as three months after operation. Gradually, the upper lip, and, finally, the eyelids take on a toned-up and straight condition, contracting when the patient swallows. The patient declares very early after the operation that the food does not lodge within the cheek, and mastication on that side is so much more satisfactory. All other symptoms, such as lacrimation, etc., disappear. The test to reaction to degeneration shows return to the normal, that is, as described before. In case many nerve fibers of the hypoglossal have been cut, or all of them, there will be a tongue paralysis, which, outside of some speech difficulty, causes no untoward symptoms.

In case the spinal accessory nerve is used, then there will be the movements of the face associated with the movements of the shoulders, unless the whole trunk is used; then, of course, there will be a shoulder droop, a condition not very desirable, and perhaps worse than the facial paralysis. Otherwise, the same course of treatment is carried out as in the hypoglossal, and the same observations are made in reference to associated function.

REASONS FOR PREFERENCES AS TO NERVES SELECTED, AND METHODS.

An end to side anastomosis is preferable to any other method because the function of the tongue is not entirely destroyed and the desired cosmetic result on the face is obtained. The cortical, as well as nuclear, centers of the facial and hypoglossal nerves are much closer to one another, and it is believed that the function of the one is more easily transferred to the other than is the case with the spinal accessory. It is believed that the associated movements between the face and tongue on swallowing are more likely to be again disassociated. Again, the act of swallowing can better be contracted than the movement of the arm; it is very embarrassing to the patient to have his face move every time the arm is moved.

It is claimed that the end to end method is preferred because more axones come in direct contact with the opposite nerve, more rapidly unite and regenerate. The spinal branch is used in preference to the hypoglossal because it is easily located and on account of its close proximity to the exit of the facial.

As to the use of the glossopharyngeal for anastomosis with the facial, I have been able to find only one reference.

REPORT OF CASES.

Case 1.—Miss H., 42 years old. Has had a suppuration of her right middle ear since childhood, when she probably had Bezold's mastoiditis, as evidenced by a scar on her neck from multiple incisions. She came to me first in June, 1905, complaining very bitterly of pains in her ear and right sided head pains, foul smelling discharge, and a crooked face which she said had been existing only for a few months. She had been coughing for some time and generally feeling bad, losing flesh, and having night sweats. One of the principal complaints was a dizziness and a tendency to fall at times toward the

right side. There was nothing important that could be elicited from the family history.

Examination.—A very frail, ill-nourished woman, weighing about 75 pounds. Temperature 99 1-2° (P. M.); pulse fair quality, and about 100. Nasal and throat findings negative. Right ear—external canal, negative, containing a fair amount of thick pus of a fetid character. After thoroughly removing it a practically normal tympanic membrane was observed, except in the lower posterior margin, where there was a small fistulous opening filled with granulations. Externally in the occipital regions marked tenderness was elicited by deep percussion on pressure. Test of hearing showed practically normal hearing through the whole range of Bezold, Edelmann, a positive Rinne. The left ear was also discharging some, but the patient knew nothing about it. There was a small perforation in the lower anterior quadrant. The hearing here was somewhat reduced and a slight negative Rinne-Weber lateralized to the left. After observation and local treatment for a short time, I decided to operate inasmuch as the patient complained more of her pains; especially when she attempted to lie on the right side of her head, she could not stand the pressure over the occipital region.

Operation.—Under general anesthesia the usual steps in radical operation were followed. When the annulus was resected and the middle ear cavity was explored, one could detect a necrotic area in the lower posterior portion of the middle ear, and that portion corresponding to the anterior extremity of the floor of the antrum, or posterior semicircular canal and the facial ridge. This area was very carefully curetted of the granulations, but I could not determine whether the semicircular canal or the facial nerve was injured at this point. I then proceeded to expose the painful area over the occipital region, and therefore made a tongue-shaped flap, with the base at the neck, or say an inverted U incision. Peeling this down, including the periosteum, we had a fearful venous hemorrhage from the region of the lateral sinus. This was controlled by compression, and on closer examination of the flap, we found a flake of bone, the outer table of the lateral sinus. This bone, however, appeared healthy. I then exposed the sinus farther back toward the torcular herophili and by compression controlled the bleeding point. On closer observation I found some granulations about this lateral sinus and a fistula.

By passing a flexible blunt pointed probe into the fistula it came out in the middle ear at the point spoken of above, namely, the lower posterior portion. Leaving this probe *in situ* I chiseled down upon it in order to transform it into an open wound. I firmly believe that the facial nerve was destroyed by the diseased process of the bone, or in any event I would very likely have injured it beyond the chance of spontaneous recovery, in order to eradicate the diseased process. The usual steps were then taken in finishing the plastic, having previously relieved the compression from the lateral sinus and tamponed the bleeding point of it. A retroauricular opening was left, in order to remove that tampon. The patient made an uneventful recovery and was perfectly free from pains and dizziness, but retained her facial paralysis. The reaction of degeneration showed plus, that is to say, no reaction resulted from the faradic current, when either applied to the nerve trunks, or muscles of the face, but there was a slow contraction of the muscles when the galvanic current was put on. That showed that the muscles were there and if union between the facial and another motor nerve took place, we could hope for results.

The photograph fig. 12 shows the condition and appearance before operation.

In the spring of 1906 the operation for facial-hypoglossal anastomosis was performed. Dr. Goldstein, St. Louis, Dr. Fleming, Los Angeles, and Dr. Owens, San Diego, California, were present and assisted. Reported in *Laryngoscope,* 1907. The operation as described in the previous chapter was performed and patient made an uneventful recovery.

Observation.—There was some soreness in swallowing and speaking which disappeared after two days. Patient declared voluntarily that there was a feeling in the right side of her face more like the other side than before operation, but this disappeared in a few days. I am inclined to believe now that that was from the reaction of inflammation in the tissues about the neck. No visible change could be noticed for about ten weeks, when a sort of a contraction of the right side of the face was noted and when patient was asked to swallow, this contraction was more apparent. From this time on, this phenomenon became more clear and gradually this tonicity was noticed over a greater portion of the face until about five months after operation; the eyelids, upper and lower, took on the tonic condition. At this time whenever the patient only thought of swallowing,

the face would move and I began to think that that would be very detrimental to the success of this operation, but the patient soon learned to disassociate these movements and at the end of the year she could swallow by very slightly moving the face. At this time the test of reaction to degeneration was practically that as is found in normal cases. There was, however, this fact shown, that whenever patient laughed or smiled the healthy side of her face would respond, whereas the paralyzed side would lag and patient would have the characteristic appearance of facial paralysis. (Fig. 13.) There never was a voluntary movement of the facial muscles without the act of swallowing. The photograph, Fig. 14, which was taken about a year after operation, shows very well the patient as recovered so far as the cosmetic purposes go.

About this time the patient's cough again became generally worse and she decided to go to the country to be out in the fresh air. She wrote to me several times that she had lost her cough and was getting along well. On December, 1907, I wrote to her to come to Chicago as I wished to present her in connection with another case, and she came. When I examined her at this time, I found her in good health and her face straight. Two days later I was notified that she was taken to the hospital with acute lobar pneumonia, under the care of Dr. J. B. Herrick.

On the 10th day of the disease the patient died. The following day I made the post-mortem examination with these findings: General emaciation of the body. The face appeared symmetrical; field of operation of ear and anastomosis showed nothing different from antemortem. Calvarium section and removal of brain. No gross pathologic changes of the brain, venous sinuses, meninges, or bony structures in the vicinity of the temporal bones within the cranial fossae. Neck dissection of nerve anastomosis. Tracing the anastomotic union of facial hypoglossal into the substance of the posterior belly of the digastric and liberating it, I found perfectly firm union of these two nerves. Examination of the right thoracic cavity showed the whole lung solid and old scars and some recent tubercular foci which were excised for histologic examination. There was some turbid fluid in the pleural cavity. Pleuritic adhesion in the postapical region. Left thoracic cavity showed some recent consolidation near the base, and an old healed tubercular apex. Heart apparently normal. Intraabdominal viscera negative.

Histologic examination of lung structure showed characteristic changes of acute lobar pneumonia, and characteristic tubercular lung disease of the apex regions.

I then excised *lege artis* the wedge of the temporal bone including the structures of the neck which demonstrated the union of the facial-hypoglossal anastomosis, and herewith present it, after carefully dissecting and preparing it, with the following findings:

The cavity of the middle ear, attic, aditus ad antrum and antrum are perfectly healed and epidermized. The posterior semicircular canal is plainly seen where it was exposed and opened, and now is healed. The region of the ostium tubae tympanicae is firmly closed off from the pharyngeal region. The resected (mastoid process) fistula from the region of the sigmoid sinus to the posterior inferior portion of the middle ear, as described in the operation, is found entirely healed. Slitting the sigmoid sinus along its course from the torcular herophili to the jugular bulb, I found it absolutely normal. The anastomosis is seen between the colored silk threads for demonstration purposes, namely, the centripetal end of the hypoglossal has a blue thread, the centrifugal end of the hypoglossal has red thread and the facial stump entering practically at right angles has a yellow thread tied about it.

Dissection of the stylo-mastoid region shows the facial nerve with yellow thread tied through it, demonstrating how very difficult it was to reach during the operation, a fact that caused me to suggest this instrument that can bluntly reach up to the stylo-mastoid foramen.

Of course, a histologic examination of the anastomosed nerves, as well as a more minute examination of the temporal bone, will follow and be of interest. This was impossible to do in order to demonstrate the gross specimen first.

Case II.—Mrs. M. This case has already been reported in the *Laryngoscope* in extenso, on account of the interesting pathologic condition of the temporal bone, so I will just briefly recapitulate in order to make clear the indication for the anastomosis.

She had an endothelioma intravascularis of the middle ear, which destroyed the continuity of structure of the facial nerve, with complete facial paralysis as a consequence. The radical removal of this tumor was not followed by any improvement of this paralysis, but there has been no recurrence of the growth

for three years, an interesting fact since these growths are supposed to be sarcomatous.

The patient hesitated for over a year and a half before submitting to the nerve anastomosis. During this time this patient suffered from a general malnutrition and marked anemia which resisted all treatment. In 1907 she decided to have the operation performed, and a test of reaction of degeneration to the facial muscles was plus and a very weak response to the galvanic current, which showed that the muscles were very weak, and if union of the nerves did take place recovery would likely be very slow.

In 1907 the facial hypoglossal operation was performed, Dr. Frank Allport and Dr. Otto Stein, Chicago, assisting. Great difficulty was encountered in finding the facial nerve inasmuch as there had been many scars in the region of the mastoid region, especially low down anteriorly from a very long after-treatment of her first operation. When the facial was finally joined to the hypoglossal, end to side, there was considerable traction in the latter nerve. Again, imbedding this union into the posterior belly of the digastric and closing up the wound. A small granuloma was curetted from the external canal of the left ear, and microscopically proved not to be endotheliomatous.

Patient made an uneventful recovery from the operation, with primary union of the skin incision. This patient made the same remarks, being a more intelligent patient than patient No. 1. She stated that her left cheek felt more natural and stronger. This also disappeared in the course of a week or two. After the patient came out of the anesthetic, I found the anterior and lateral portions of the left side of her tongue paralyzed, none of which was evidenced in Case 1. During the first six months following the operation, during which time the patient received daily electric massage and tonic treatments, there was very little improvement seen; in fact, so far as appearances go, only a beginning crease, such as one sees as a natural contracture following the paralyzed muscles is observed, and only to be differentiated when the patient swallowed. From this time on the improvement was more in the upper branches; she could close her eyes better, and on the application of the faradic current, small contractions followed. However, I want to put this case on record up to the present day, not as a cure, but to prove the point that when

one has a positive test of reaction to degeneration, and clinically has strong evidences that the nerve is very likely severed for a greater distance and scars, tumors, sequestra, or other foreign bodies, interposed between its ends, one should do the anastomosis as quickly as possible and not wait for nature to bridge it over. Another point is the general condition of this patient, namely, the anemia.

Case III.—Mr. M. This case has already been reported in connection with the report on Bezold's mastoiditis in *Journal of Ophthalmology and Oto-Laryngology,* 1907, and I will therefore omit the detailed history. Suffice it to say at this time, that the man had a permanent facial paralysis associated with a marked necrosis of the temporal bone along the greater course of the facial nerve, including the nerve. On August 4, 1907, I made a facial-hypoglossal anastomosis, only that the technic differed in making it end to end. Dr. Ray, of Louisville, and Weaver, of Sherman, Texas, assisted. There was again a great deal of difficulty in dissecting the facial, owing to many old scars on the neck from the Bezold mastoiditis many years before. We found quite a number of enlarged lymph glands, submaxillary, over the region of the hypoglossal that required dissection and took considerable extra time.

Observations.—After the patient came out of the anesthetic, he had complete paralysis of the left side of his tongue, and there was considerable difficulty in speaking and swallowing. This man had no such feeling of the paralyzed side of his face as in Cases I and II, after operation—that is, primary tonicity. Ten days after the operation, when the stitches were removed, there was found a small fistula in the region of the parotid gland, discharging saliva, and when the patient would suck a lemon, this flow would be markedly increased. This fistula was very likely produced during the manipulations to dissect the facial nerve by a sharp retractor. This precaution against such an accident is brought out by Murphy.

As to the course of this case, I am not able to report any further than that he left Chicago about one month after the operation, having had no after-treatment. Being a laborer and in very bad circumstances, he went to Cleveland to seek employment. About four months after the operation I received a letter from Dr. Large, who chanced to see him in his clinic, and he states that the face appears completely paralyzed,

and the tongue paralysis is giving the patient considerable trouble. No conclusion can be drawn from this report, however, as a test for reaction of degeneration, and the observation of one who has seen the case before operation are necessary.*

Case IV.—Mrs. S., 32 years old. Suffered for a long time with tic doloroux and facial paralysis on the left side. When I first saw her she had received every possible treatment for the cure of her tic, including the injection of osmic acid (Murphy); neurexeresis of the three branches and resection of the Gasserian ganglion (by Carl Beck) without positive relief. Examination of the ear showed no evidences of trouble there, and the test of reaction to degeneration was negative. The eyelids and forehead muscles were not affected. The eye could not rotate outwardly, and there was a paralytic squint. She could not hear in this ear. Therefore, I made a diagnosis of some central pathologic lesion involving the fifth, sixth, seventh and eighth nerves, probably tubercular, as there had been a history of this affection.

On January 7th, 1907, I operated, reopening the original bone flap that had been made by Dr. Carl Beck in operating for the Gasserian ganglion, and found a very markedly thickened dura. (Sectioned later and found just granulations, extradural, but no tuberculosis.) Elevating the brain structure I found considerable matting together of the base of the brain through the dura. I explored the region of the exit of the above nerves from the brain and did not find any gross lesion. Knowing that exploring this region of the brain structure would very likely kill the patient, I stopped the operation. However, before closing up I explored the brain from above in various directions and found nothing in the shape of a tumor. To my astonishment the patient recovered and was free from the neuralgic pains. Enthusiasm on my part and the desire of the patient to have her face straightened, I agreed to do a facial

*Just before going to print, patient, Chas. M., presented himself, after an absence of several months (during which time he had been under the care of Dr. Large), showing a complete recovery from his paralysis. Not only has the tone and associated function with swallowing returned, but the patient also has actual voluntary movement of the formerly paralyzed side of the face. This is the first case I have seen to have this phenomenon. I also wish to state that this patient has had no after-treatment, as electricity, etc., and has lived in the poorest hygienic surroundings.

hypoglossal anastomosis. The hypoglossal nerve was of course not involved in the above intracranial trouble. On February 5, 1907, I operated in the usual way, intending to do the end to side procedure. Dr. Vaughan, Chicago, and Dr. Edwards, of Kansas, assisted When I had the facial dissected and excised from the stylomastoid foramen and attempted to bring it into close proximity to unite with the hypoglossal, I found it impossible. Having just read of Gluck's experiments to use living tissues as conductors for nerve fibers to regenerate, I used a neighboring large branch of the superficial cervical nerves. Excising a long enough piece, I made with it an end to end procedure with the facial, and implanted it into a slit of the hypoglossal as usual. This whole area was covered by a layer of the sterno-cleido-mastoid muscle, that being nearest at hand, to prevent adhesions. Patient recovered from this operation without any untoward symptoms. Shortly after, however, nephritis was diagnosed and a general anasarca followed. The patient did not live more than two months after the performance of this last operation, and consequently I can add nothing further to the subject. There was never any statement made by the patient that she felt any primary tone on the operated side of the face, and no evidences of recovery were demonstrable. The patient had some trouble in swallowing and the tongue lagged considerably on the left side.

Inasmuch as a post-mortem was not permitted, I cannot say what the conditions of the anastomotic points were, nor can I possibly find out the specific etiologic factor.

Case V.—Mr. W. B. D., Louisville, Ky., aged 53 years. Has always enjoyed good health until he developed an earache in right ear, which was diagnosed as an abscess by Dr. Cheatam, who lanced it and applied leeches, but without relief. Pains did not diminish. He was taken to infirmary after two days' time and drum again incised. Hearing was never much interfered with and there was never much soreness back of ear. Deep-seated pain over right eye. Expectant treatment was instituted, consisting or repeated irrigation of the ear with hot water and lying on hot water bag; this was continued for six weeks, when treatment was changed to flaxseed poultice every 20 minutes for about 72 hours. After elapse of one day Dr. Cheatam performed mastoid operation, following which there was no relief from pain. Six days later second operation was performed by Dr. Cheatam, assisted by Dr. Ray, following

which the patient still was not relieved from pain and eye aching. Immediately after recovery from anesthesia facial paralysis was observed.

Findings at time of operation given by Dr. Cheatam and Dr. Ray are as follows:

These deep-seated head and eye pains continued for about three months, gradually abating until they disappeared. The healing of wound back of ear required a considerable length of time, about five months. The discharge from the middle ear ceased a week or ten days after, never recurring. The facial paralysis had never taken on any change in spite of all local and general treatment. Besides difficulty in speech and cosmetic appearance there was a metallic taste in the anterior and lateral half of tongue (right side), which was constant, especially when he cleared his throat. Lacrymation considerable. He did not use right side of face for chewing, as the food lodged in the cheek cavity. Patient thought that salivary secretion and taste might have diminished some. Olfaction did not seem to have been affected. Breathing (nasal) as good as ever. General health has been good except he had some trouble with bladder with bloody urine, which would clear up and return. There was some difficulty in urination, sometimes burning. Examination of urine was made by Dr. Wendell, Dr. Wathen, Jr., Dr. H. Koehler, and kidneys found normal, but there had been no general examination made since operation. Appetite good, patient sleeps well, bowels regular. Habits are, practically abstainer.

Examination.—Characteristic facial paralysis. There was primary contracture at lower lip extending to the margin of lower jaw. Lower lid drooped so as to evert lacrymal duct down and out, showing accumulation of tears constantly in lower sac. The alae of nose, right side drooping, narrowing nostril. Upper lip hung over margin of upper teeth, and lower lip, caused by upper lip, held closer to lip margin. At back of the ear was found evidence of an incision leading from a line drawn across the upper margin of auricle downwards to about an eighth of an inch from the tip of lobule, in middle of which there was a depression in the mastoid process of the size of a lima bean. In the upper margin of incision there was a keloid scar firmly adherent to bone.

Patient was observed last October under superficial examina-

tion, but a positive examination made as to a reaction degeneration, which herewith is repeated:

Test of Reaction of Degeneration.—No response to the faradic current. When galvanic current is applied patient feels contractions at make and break, but not observed by myself.

Nasal Examination.—Practically negative with a few irregularities.

Examination of Tongue.—Perfectly straight.

Examination of Palate.—Shows absolute movement normal, the muscle of uvula reacting promptly.

Examination of Right Ear.—Stenotic canal (externally). The tympanic membrane seems to be entirely regenerated. No evidence of disease in remaining portion of canal.

Examination of Hearing.—Hears highest tones. Hears low tuning fork. Weber laterates to right. Rinne negative. Shorter on right side. Rinne positive on left side.

General Examination.—Revealed no pathologic condition as to his kidneys, gastro-intestinal tract and heart and lungs. February 12, 1908, facial-hypoglossal anastomosis was performed, Dr. Ewing M. Day, Pittsburgh, Dr. J. Pratt, Shelbyville, Ky., Dr. L. J. Hughes, of Chicago, assisting. I found the facial nerve unusually easy in this case, for in the former cases this procedure lasted very long. On account of the long continued drainage after the mastoid operation, I expected much trouble from adhesions. We made the end to side implanation and buried the nerve in the posterior belly of digastric. Time of operation 2 hours and 48 minutes.

Observations.—With the exception of a marked nausea, which he said he had also with his other operations, the patient recovered very well and continued to remain normal for five days, when his temperature rose to 103.4°. Examination of the wound showed a marked swelling in the region of the parotid and along the lower jaw. Releasing some of the upper sutures a flow of saliva of established (salivary fistula), and on the following morning he had a temperature of 99°. Every day following until this day, February 21, 1908, his temperature fluctuated between 99° and 102°, with some degree of uncomfortable feeling. Examination of the pharynx shows a small degree of edema of the uvula. There can be no further statements made at this early time as to results.

CONCLUSIONS.

The fact that one case out of a dozen of facial-hypoglossal anastomosis will recover is sufficient encouragement and indication to justify its performance.

In order not to bring this procedure into disfavor, one should select his cases and this selection should be based upon a thorough examination, especially as to the reaction of degeneration. That is to say, the muscles must be present, no matter how flaccid, and the galvanic electric current will bring that out, as stated above.

A thorough knowledge of the anatomy and technic is absolutely necessary and I know of nothing better to recommend than practice suturing of nerves on dogs and perform the operation on the cadaver two or three times before doing the operation.

One should not be satisfied by simply doing the operation, but great care should be exercised in the long, continuous after-treatment of electricity, massage, etc.

Since the reading of this paper, the author has operated on the sixth case which has the following history:

Mr. B., 18 years of age, referred to me by Dr. Casey Wood, when 1½ years old had a marked mastoid disease with secondary abscesses in the neck, anterior and posterior, as well as anterior to the auricle in the parotid regions of the left side. Dr. Barkan, of San Francisco, operated at the time, and it required a great length of time before boy recovered. His face, previous to the mastoid operation, was straight. Following the procedure he had complete facial paralysis.

Examination.—Characteristic picture of the condition with all the usual symptoms. A number of large scars are found on his face, neck and back of the ear. In this locality is quite a large fistula, communicating with the auditory canal, but it is entirely dry. Test of reaction to degeneration shows it is negative, that is, there is no response to any kind of current, and consequently a case with very poor prospects of recovery. In fact, from the length of time that the paralysis has existed one would not expect any result and is a contraindication to the operation.

I wish, however, to state, that as this is the son of well-to-do parents, who took advantage of everything known to them to aid the boy in recovering function in the paralyzed side of his face, the patient has, all these years had stimulating treatments

to the nerves and muscles of the face. Consequently there was some chance that remnants of muscles might be present.

Dr. Casey Wood and the father decided, nevertheless, to have me try to do an anastomosis and take a chance on the result.

Operation.—Through one of the existing neck scars I soon reached the region of the facial nerve. To my great disappointment I found the complete absence of any such structure, and, if it was present at all, it was not recognizable as a facial nerve, nothing resembling a nerve structure in the whole course. I dissected towards the face and here also found no trace of any of the branches. Consequently I closed up the wound and stated to the parents the absence of the facial nerve. The young man made an uneventful recovery from the operation.

Two weeks later I performed a plastic on his face in the following manner: Through a scar situated over the zygomatic region which was first resected, a long straight needle armed with strong silk was passed well under the skin towards the labial fold, there made a loop, and needle again made to come through the original entrance opening. Drawing the thread taut, somewhat puckering the skin so as to make an artificial labial fold, the threads are tied and the wound closed by primary intention. Patient recovered with a very good cosmetic result, as the accompanying pictures show.

Case IV.—Mr. M. has reported since the reading of the paper and sent a photograph. It shows distinctly a formation of labial fold and he reports associated movements of his face with the act of swallowing. The tone of the paralyzed side has much improved.

Case V.—Mr. D. Doctor Moren, of Louisville, reports that the muscles of his paralyzed side of his face are responding much better and patient declares an improvement in the tonicity of his face.

Case II.—Mrs. M. has an improvement in her facial expression but her general condition of anemia is constantly increasing and the chances of recovery are not very good.

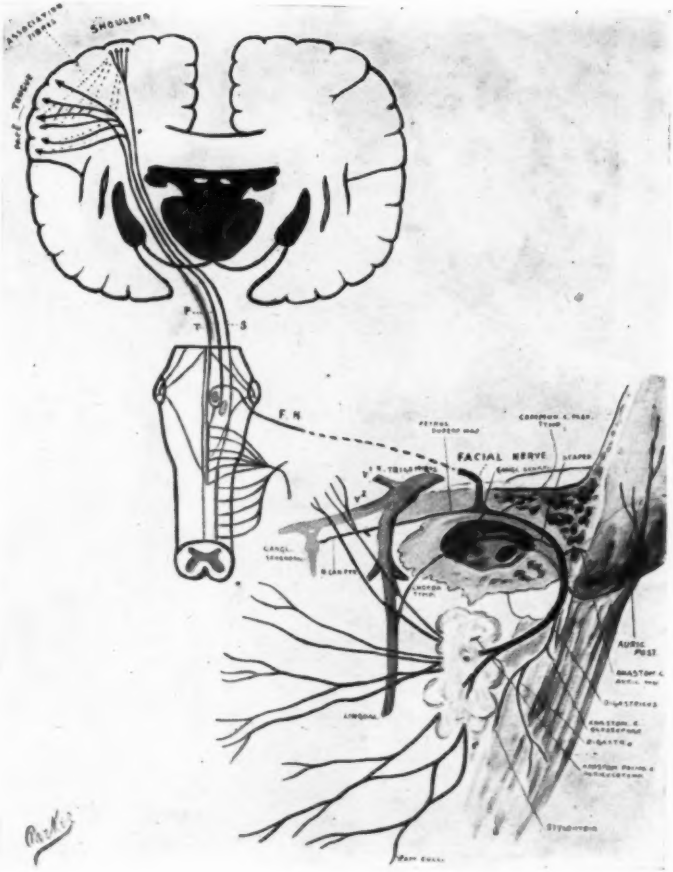

FIG. 1.

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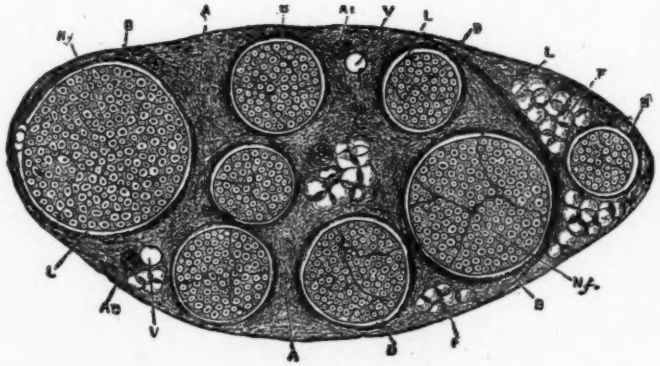

Fig. 2.—Transverse section of the sciatic nerve of a cat about x 100.—It consists of bundles (Funiculi) of nerve-fibres ensheathed in a fibrous supporting capsule, epineurium, A; each bundle has a special sheath (not sufficiently marked out from the epineurium in the figure) or perineurium B; the nerve-fibres Nf are separated from one another by endoneurium; L, lymph spaces; Ar, artery; V, vein; F, fat. Somewhat diagrammatic. (V. D. Harris.)

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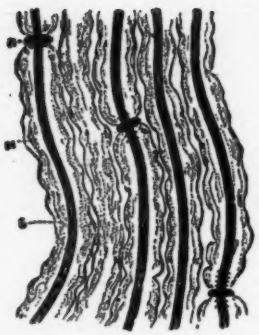

Fig. 3.—Several fibres of a bundle of medullated nerve-fibres acted upon by silver nitrate to show peculiar behavior of nodes of Ranvier, N, toward this reagent. The silver has penetrated at the nodes, and has stained the axis-cylinder, M, for a short distance. S, the white substance. (Klein and Noble Smith.)

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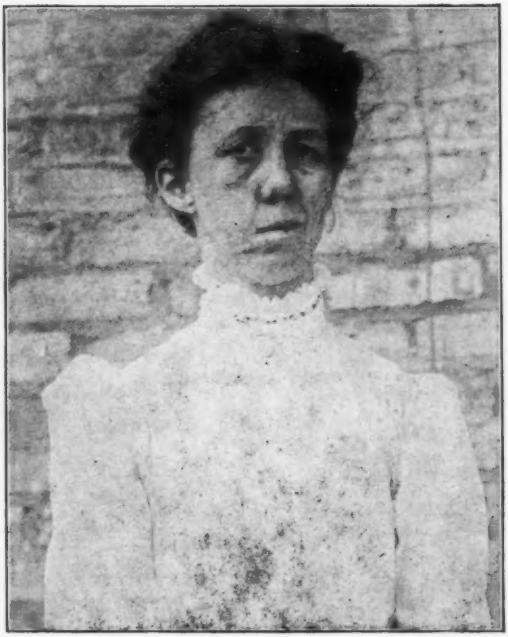

Fig. 4.

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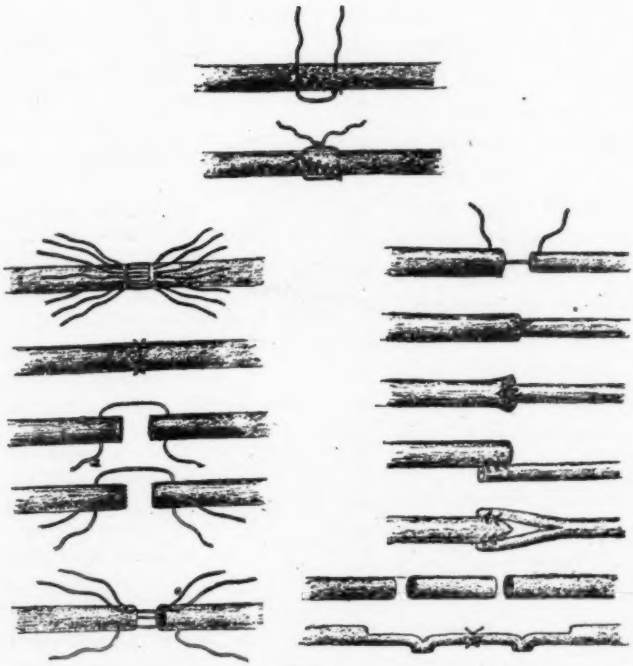

Fig. 5.

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FIG. 6.

Fig. 6—Represents the surgical anatomy of the field of operation of the facial nerve with the other two nerves (spinal accessory and hypoglansal).

1. Sternocleidomastoid artery.
2. Facial nerve.
3. Occipital branch of posterior auricular artery.
4. Posterior auricular branch of facial nerve.
5. Mastoid tip.
6. Sterno mastoid, muscle cut and retracted.
7. Lesser occipital nerve.
8. ii Cervical nerve (ventral Br).
9. Accessory nerve.
10. Accessory nerve.
11. iii Cervical nerve (ventral br).
12. iv Cervical nerve (ventral br).
13. Brachial plexus.
14. Ascending branch of transverse cervical.
15. Trapezius.
16. Omohyoidens.
17. Descending branch of transverse cervical artery.
18. Subclavian artery.
19. Subclavian vein.
20. Pectoralis minor.
21. Deltoidens.
22. Cephlic vein.
23. Thoracs acromial vessels.
24. Clavicular portion of pectoralis major.
25. Ant. Thoracic nerve.
26. Clavacular port. of pectoralis major.
27. Sternocleidomastoidens.
28. Sternocleidomastoidens.
29. Sternocleidomastoidens.
30. Innominate vein.
31. Vagus nerve.
32. Com. cartoid nerve.
33. Inf. bulb of int. jugular vein.
34. Phrenic nerve.
35. Super. cervical artery.
36. Thyroid glands.
37. Ansa Hypoglossi.
38. Descending br. of hypoglossi.
39. Ascending br. of cervical artery.
40. Superior thyroid artery.
41. Descending br. of hypoglossal.
42. Superior laryngeal artery.
43. Ext. caroted artery.
44. Thyreohyoid br. of hypogl.
45. Lingual artery.
46. Hypoglossal nerve.
47. Stylohyoidens.
48. Digastricus.
49. Submental artery.
50. Mylohyoid nerve.
51. Submaxillary gland.
52. Ant. facial vein.
53. Paroted gland.
54. Posterior facial vein.

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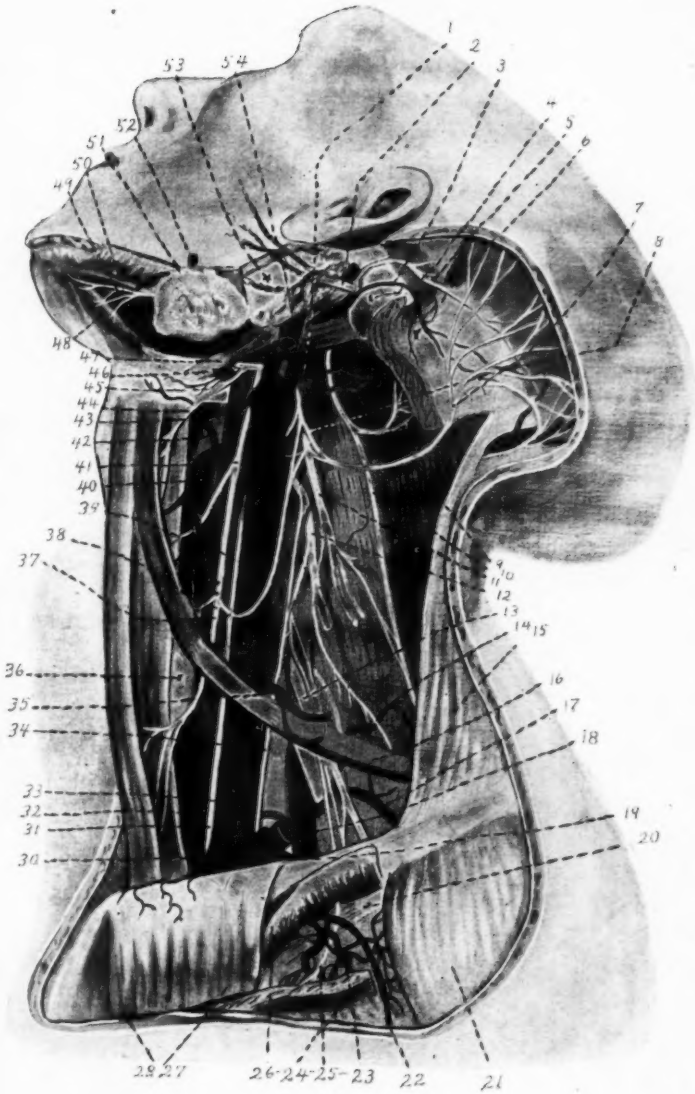

FIG. 6.

See next page.

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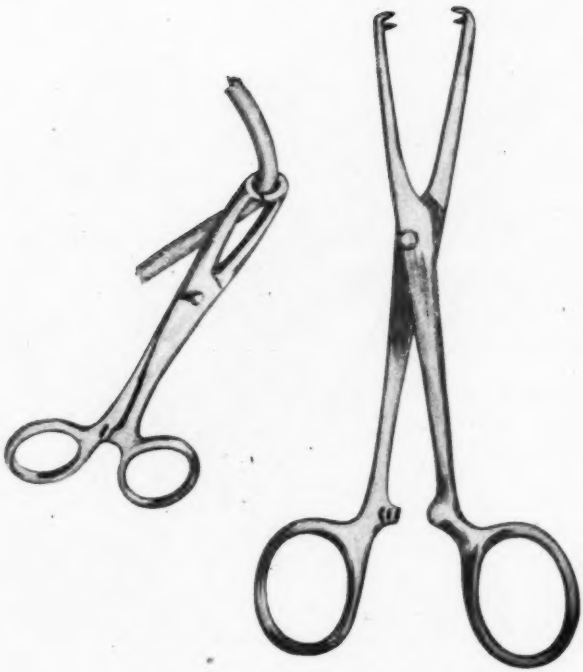

Fig. 7.

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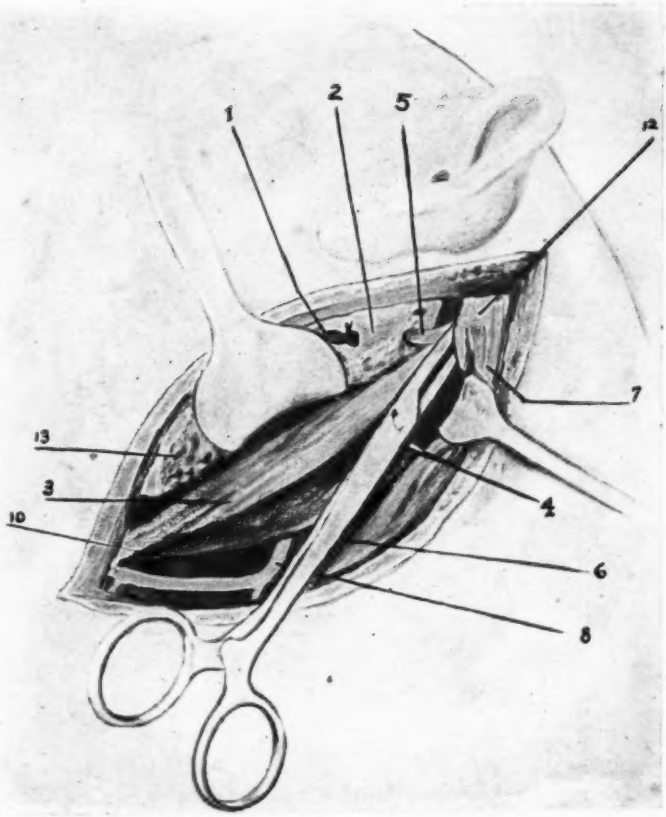

FIG. 8.

Illustrating the instruments in place for dissecting the facial nerve into the depths of the stylomastoid foramen.

1. Ant. facisl vein.
2. Parotid gland.
3. Stylohyoidens.
4. Post. belly of digastricus.
5. Facial nerve cut.
6. Int. jugular vein.
7. Sternocleidomastoidens.
8. Hypoglossal nerve.
10. Common caroted artery.
12. Mastoid tip.
13. Submaxillary gland.

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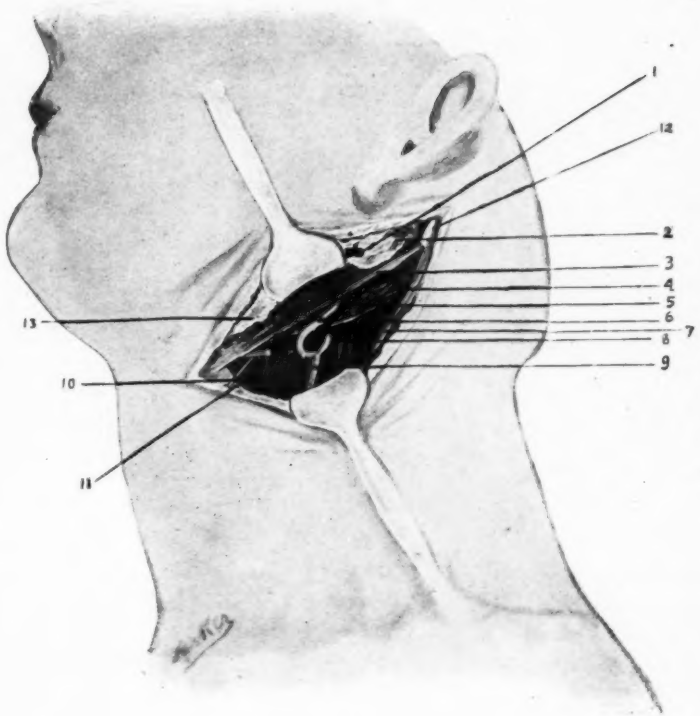

FIG. 9.

Fig. 9. Representing the facial hypoglossal anastomosis by the end to end method.

1. Anter. facial vein.
12. Mastoid tip.
2. Parotid gland.
3. Stylohyoidens muscle.
4. Post. belly of digastric M.
5. Facial nerve (cut off at stylo-mastoid foramen and turned down under stylo-hyoidens, implanted into No. 11).
6. Internal jugular vein.
7. Sternocleidomastoidens muscle.
8. Hypoglossal nerve.
9. Hypoglossal nerve, its descending branch.
11. Hypoglossal nerve (with the facial stump implanted).
10. Common carotid artery and its subdivisions.
13. Submaxillary gland.

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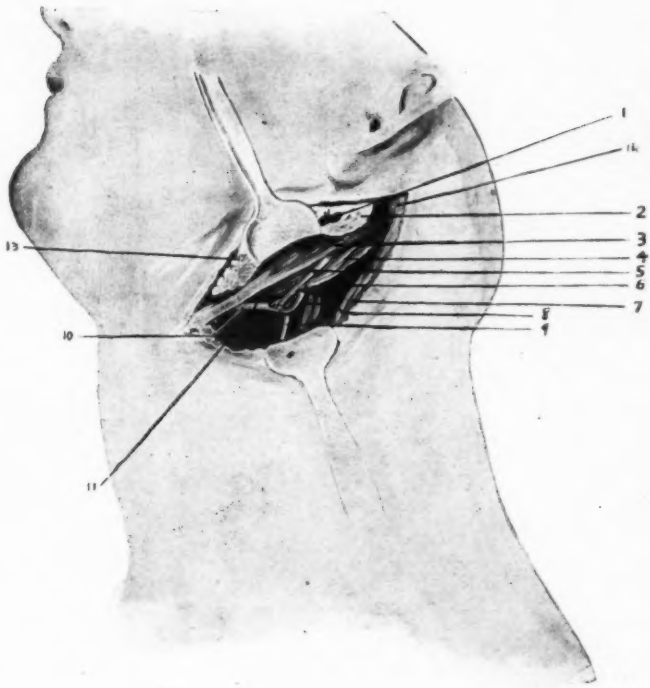

FIG. 10.

Representing the facial hypoglossal anastomosis by the end to side method.

1. Ant. facial vein.
2. Parotid gland.
3. Stylohyoidens.
4. Post. belly of digastricus.
5. Facial nerve cut and pulled under the stylohyoidens.
6. Internal jugular vein.
7. Sternocleidomastoidens.
8. Hypoglossal nerve.
9. Hypoglossal nerve, descending branch.
10. Common carotid artery.
11. Hypoglassal nerve.
12. Mastoid tip.
13. Submaxillary gland.

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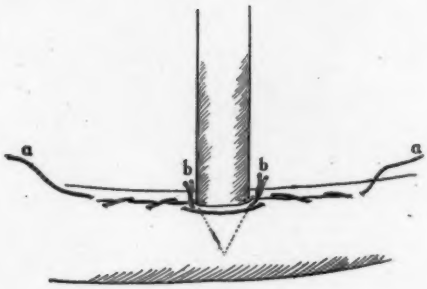

Fig. 11.

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Fig. 12.

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Fig. 13.

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Fig. 14.

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X.

THE OPERATION FOR THROMBOSIS OF THE SIGMOID SINUS AND INTERNAL JUGULAR VEIN, OF OTITIC ORIGIN.*

By Frank Allport, M. D.,

Chicago.

One of the most important sequelae of middle ear infections is phlebitis or thrombosis of some of the venous drains of the head, and particularly when such pathologic conditions occur, as they usually do, in the sigmoid bend of the lateral sinus. Disease of this nature is found not only in the sigmoid sinus, but also in the lateral sinus proper, the superior and inferior petrosal sinuses, the cavernous sinus, the jugular bulb, the internal jugular vein and its connections, etc. The sigmoid sinus is, however, its favorite location on account of its vascular connections with the tympanic cavity and adjoining cells and its close contiguity to these frequently infected structures. As the jugular bulb is located directly beneath the tympanic cavity, an unusually high dome to the bulb, or necrosis of the tympanic floor, or congenital dehiscences in the floor, will assist in the conveyance of infections. This will occur through the vessels and lymphatics connecting the tympanic cavity with the bulb, and may produce a primary phlebitis or thrombosis of the bulb, which has not proceeded through the usual channels of the antrum, mastoid cells and lateral sinus. These conditions are usually found in children, develop rapidly, and generally demand immediate operation with excision of the internal jugular vein. I do not believe that actual intravascular phlebitis and thrombosis of the sinus and vein occur as frequently as might be supposed from a perusal of much of the available literature of the day, because a fairly large experience in mastoid work for many years has not disclosed this to be the case. Nevertheless, the disease occurs with sufficient frequency to demand the most careful study in order

*Read before the Middle Section of the American Laryngological, Rhinological and Otological Society, Indianapolis, February 22, 1908. From the Journal of the A. M. A.

that prompt and skillful action may be taken whenever its existence is actually disclosed.

RISK IN SINUS OPERATIONS.

The surgery of otitic phlebitis and thrombosis has made great progress of late years and many lives have been saved thereby that must otherwise have been inevitably lost. I fear, however, that over-enthusiasm along these lines may have led some thoroughly competent and trustworthy observers to overestimate the frequency and importance of the subject and to underestimate the dangers of operative interference. The opening of the sigmoid sinus, for instance (first seriously proposed by Zaufal in 1880), with its possibilities for infection, especially when done through an already infected mastoid cavity (which is, of course, usually the case), is an operative procedure of importance and danger. Yet some surgeons practically advocate its performance for diagnostic purposes where any reasonable suspicion points to an intrasinus involvement. In my opinion the sinus should usually be opened in the face of mastoid disease, either acute or chronic, accompanied by characteristic phlebitic symptoms, such as chills, sweats and wide excursions of temperature and pulse. It should also be done in the presence of a virulent infection, especially of the streptococcus type, a significant differential blood test, etc. Nevertheless, I have seen a number of such cases cured by the removal of necrosed bone, granulations[1] and pus from the sinus neighborhood. I should, therefore, be inclined to wait a few hours after the removal of such débris, before opening the sinus itself, unless the patient's condition and symptoms are reasonably decisive and urgent in character, and unless an inspection of the exposed sinus renders the presence of a thrombus exceedingly probable.

It must have been the experience of most surgeons that phlebitic symptoms frequently and permanently may be relieved by a removal of neighboring necrosis, granulations, pus, etc., which perpetually infect and reinfect the sinus walls. This fact is especially emphasized by Bergman in his writings and can certainly be substantiated by all operators of experience. In this observation lies an earnest warning against too

1. The existence of granulations on the soft sinus wall is regarded by many as beneficial, in that it is nature's obstruction to the invasion of microorganisms, and many surgeons carefully refrain from disturbing them during the course of an operation.

energetic operative procedures, without quite positive indications. It is sometimes the case that septic symptoms following a mastoid operation are due to pneumonia, typhoid, malaria and other coincident conditions which should be duly considered and properly weighed before proceeding to a sinus operation. It should not be forgotten that, while lives may occasionally be sacrificed by proper conservatism, it is probable that more lives would be lost by injudicious and hasty surgery.

The provisional determination to open the sigmoid sinus is usually formed before the mastoid has been operated on, or after an operation has been performed and several days have perhaps elapsed, during which time the condition of the patient has not materially improved. This resolution is, of course, liable to be changed by existing intramastoid conditions, after the mastoid cortex has been removed, at the time of the primary operation. In all cases where sinus symptoms are clearly present, the bony covering should be freely and carefully removed and the soft sinus exposed for inspection, whether the osseous partition is necrosed or not. Should the inspection reveal an apparently healthy sinus, operative procedures may be suspended (unless the patient's condition is too grave), as sharp sinus symptoms are sometimes produced by a beginning absorption or phlebitis. This may originate in an infected mastoid cavity, relief from which is not infrequently noticed after a thorough exposure and cleaning of this space.

After this has been accomplished and the patient carefully watched for some hours or perhaps one, two or three days, and the sinus symptoms remain the same or grow worse, the sinus should be unhesitatingly opened, even if its local appearances are apparently healthy. Much the same procedures should obtain in case necrosis, granulations and pus are found in the sinus region, for such pathologic products continually bathing the soft tissue tend to become absorbed and to produce the characteristic danger signals. Such debris may be carefully removed and the sinus amply inspected. Should it be found of healthy appearance, one may stay operative procedures a little, unless the symptoms are quite urgent. Many patients have recovered and will recover under such conservative management, and this fact points a significant lesson.

THE APPEARANCE OF THE UNCOVERED SINUS.

Of course, the entire subject of opening the sinus hinges

on the probable existence or non-existence of a thrombus, and this cannot always be easily determined. A discussion of this topic is too prolonged for the present article, and is apart from the specific subject under consideration.[2] As the physical appearance of the uncovered sinus, however, must be observed while operating on a patient suffering with sinus symptoms, this phase of the subject may with propriety be here considered, and it may be said that even a physical examination of the sinus does not always clear up the pathology. The sinus is usually bluish-white in color and easily compressible. Nevertheless, the neighboring necrotic bone, etc., may stain and darken the outer coats of the vessel, without the existence of any intramural clot, or a perfectly normal color may characterize a sinus, in whose interior a thrombus may be found.

A sinus may be thoroughly compressible and yet contain a parietal clot, where the obstruction is not yet complete, but a solid, resisting sinus is, of course, a reasonably positive sign of an obstructive clot. The emission of pus from the mastoid foramen is a significant symptom, as it may then be assumed that extradural pus is present in the cerebellar fossa, around the sigmoid groove. I am opposed to the insertion of an aspirating needle into the sinus in order to ascertain the fluidity of its contents. If nothing is withdrawn it is safe to assume the presence of a thrombus, but blood may escape in case of a partially organized clot, and if the vessel is healthy the operator may easily regret having punctured and probably infected a normal sinus; if the sinus is to be opened at all, it is much better to make a complete incision rather than a puncture. The stroking and compression test of Whiting cannot be commended, for, while it may facilitate the diagnosis, it can easily displace infected thrombotic particles and set them into circulation with disastrous results.

THE PROBLEM OF DIAGNOSIS.

It will thus be seen that making a diagnosis in cases of otitic sinus thrombosis is beset with some difficulties and perplexities. Yet a correct conclusion can usually be reached,

2. A tolerably complete analysis of the symptomatology of sinus thrombosis, etc., can be found in a communication by the writer in the Chicago Medical Recorder, January, 1906, and in other books and periodicals.

especially when a consideration of the general subjective and objective symptoms has been aided by an operative exposure of the sinus itself through the mastoid cavity. Fortunately this (mastoid operation) is a procedure which should be performed primarily and, therefore, nothing is lost and everything gained when a mastoid operation is advised in a suspected otitic sinus thrombosis, for the cells, etc., may be thoroughly opened and cleaned and the sinus inspected and perhaps opened during one operative procedure.

Should an operation, or at least an inspection of the sigmoid sinus be decided on, in a case of otitic origin, it is only necessary to consider the operative steps in connection with a thorough exposure of the mastoid cavity, for this is at the same time the easiest as well as the best avenue of approach. The mastoid operation should first be completed in all its details, for, in case troublesome hemorrhage occurs, it might be extremely difficult to continue further operative procedures on the bone. In addition, the work on the sinus should be prosecuted under the most aseptic conditions possible, and this can be best accomplished after the mastoid interior has been thoroughly cleaned. As has been previously said, the bony covering of the sinus may be in a necrotic, softened and fistulous condition, surrounded by granulations and bathed with pus.

THE REMOVAL OF GRANULATIONS.

The removal of such pathologic products can be accomplished with comparative ease by curette, chisel, bur or rongeur, extreme care being exercised in all such manipulations to avoid injuring the soft dural covering of the vessel. Some authors advocate the non-removal of granulations, believing them to be Nature's obstruction to the invasion of the sinus interior by microorganisms. However this may be in theory, I believe that such tissue is best thoroughly removed, not only to place the parts in as clean a condition as possible, but also to facilitate a careful examination of the sinus, which cannot be satisfactorily accomplished through a granular mass. In most instances I accomplish this work by means of a strong, narrow rongeur, one blade of which is inserted inside and the other outside of the bony sulcus, being careful to avoid pressure on the underlying vessel and only removing small fragments of bone at a time. Objections have been made that the slight pressure on the sinus may induce a fracture of its possibly

thrombotic contents, which will be swept into the circulation and induce migratory infection. This objection seems like a refinement of technic and a magnifying of possibilities, for, while such an accident might possibly occur, it is altogether unlikely. It certainly would seem to be a better instrument than the chisel, with its possibilities of bone fracturing and splintering, with perhaps a sinus puncture and an embarrassing hemorrhage.

All the necrosed bone should be removed, no matter how far it extends, either in the direction of the torcular or the bulb, and it is well to remove even some healthy bone in order to insure complete elimination of all necrosis (Fig. 1). The sinus should then be minutely inspected, and if its opening is determined on the operation should be continued after the parts have been thoroughly cleaned and irrigated with antiseptic solution. If it is deemed best to await developments the wound should be cleaned, irrigated and dressed as after other mastoid operations.

In case a primary opening of the sinus is determined on either at the original mastoid operation or at a later date, it should be remembered that an ample exposure is absolutely essential to correct surgical manipulations. The sinus must be exposed, therefore, for at least a short distance above its knee and as far downward in the direction of the jugular bulb as possible. This free exposure may be accomplished by a removal of the necrosed bone alone, where such necrosis is extensive in character, or it may be necessary to remove considerable healthy bone in order to render the sinus easily accessible.

LOCATION OF THE SINUS.

Unless the situation of the sinus is indicated by a fistula, its position is uncertain. In children it is located more superficially and further forward than in adults, and even in the latter its location is by no means fixed. It is usually, however, to be found at a point about one-half an inch behind the posterior bony meatal wall, just back of the antrum, and a thorough mastoid operation will usually disclose its convexed outlines. The sinus should first be exposed by chisel, bur or curette, after which the bone is best removed with a strong, delicate and narrow rongeur, as previously described. The bone should be carefully removed in small pieces and the operative field examined after each bite of the forceps, care being taken not

to wound the sinus, either by perforating its covering by a sharp fragment of bone or by rupturing an already softened dura by carelessly pulling away adherent osseous chips. The latter accident may be averted by, from time to time, passing a small spatula between the soft sinus and its bony covering and gently separating the two.

After the sinus has been fully uncovered all debris, etc., should be thoroughly removed and the operative field abundantly flushed with antiseptic. The hands and instruments of the operator should be recleaned and every effort made to prevent sinus infection. In opening the sinus there is danger of a fatal aspiration of air occurring, to prevent which the patient's head should be dropped low and the operative area flooded with sterile water. The sinus should now be opened by a free incision along its length, being careful not to pass through both walls of the sinus into the cranial cavity, an accident which is by no means impossible. If there is a flow of blood, two tight rolls of gauze or cotton held in fixation forceps (which should be previously prepared) should be placed one above and the other below the incision, remembering that brain substance and not bone lies beneath the sinus, and that the amount of pressure should, therefore, be limited (Fig. 2). Such pads may be held by the operator, or by his assistant, or both operator and assistant may each hold one; they should not be applied, however, until after the sinus has been opened and an opportunity has been given for a burst of hemorrhage to occur and possible thrombotic particles to become expelled. These pads may be lifted alternately and accurate information obtained as to whether the hemorrhage occurs from above or below.

If free hemorrhage occurs from both directions it is proper to assume the absence of a clot, or at least that any existing loose clots have been expelled by the force of the escaping blood. If the hemorrhage is slight or absent from either direction, a clot in that direction is to be assumed. If the vessel is assumed to be healthy, some iodoform gauze, rolled into a cylinder long enough to cover completely the sinus incision, should be firmly placed over the vessel and supplemented by layer after layer of gauze carefully held in place. The other portions of the mastoid cavity should then be packed with gauze and the entire mass of packing held in position by a firmly applied bandage which may usually remain in position

for three, four or five days, when it should be carefully soaked off (to prevent hemorrhage) and more packing and bandage applied.

The danger of hemorrhage is usually past by the time the second or third dressing is done. In case there is hemorrhage from below and not from above, the sinus should be further incised in the direction of the torcular, and more of the bony sulcus removed if necessary, in order that adequate room for further operative procedures should be afforded. In considering thrombi, which extend from the sigmoid sinus back toward the torcular, it should not be forgotten that clots extending in that direction are not usually so infective in character as those spreading in the direction of the bulb. The venous current, of course, naturally flows from the head downward, and, while infectious particles may easily pass down with the blood current, they do not so easily pass upward and backward against the current. It should not be forgotten, however, that blood currents in valveless veins seek the direction of least resistance and may easily become reversed on contact with intravenous obstructions. It is, therefore, quite possible that, while a thrombus in the immediate vicinity of the antrum may be highly infectious in character, as it extends backward toward the torcular it may gradually become perfectly innocent, and composed of benign accretions of normal blood clots, that form a natural and healthy obstruction to the passage of infectious microorganisms.

TREATMENT OF THE THROMBUS.

It is, therefore, questionable just how much such a thrombus should be disturbed, and some authors strongly advocate leaving it alone. I believe that if the clot is hard and apparently extensive, it should be gently curetted at its visible portion, in order to remove its infectious layers, and then sprinkled and packed with iodoform powder and gauze. In case, however, the clot is small, or soft and unhealthy in its appearance, it should be completely removed by the curette until free hemorrhage occurs (it being remembered that quite free hemorrhage may occur, even with a considerable parietal thrombus, or from, for instance, the superior petrosal sinus, though the sigmoid sinus is more or less blocked with thrombotic accretions) even if it becomes necessary to remove more and more of the bony sulcus with a corresponding extension of the sinus

incision. A small-sized curette should be employed for the removal of thrombi, small enough to allow of intrasinus manipulations, and its spoon may, of course, be carried considerably beyond the limits of the bony opening (Fig. 3).

It should not be forgotten that a sinus infection of several days' duration may soften the sinus walls to such an extent as to render their perforation by a curette exceedingly easy; curetting should, therefore, be carefully performed, lest brain infection be thus produced. Ease of manipulation may be much facilitated in all sinus curettements by not only incising the sinus, but by excising a large piece of its outer dural wall, and diagnosis and surgical precision will be greatly aided by exposing the intrasinus openings of the mastoid emissary vein and the superior petrosal sinus. The former will be found in the posterior wall of the sinus at a point about on a level with the floor of the ordinary radical mastoid operation when finished. The latter enters the sigmoid sinus from the cavernous sinus at a point about on a level with the roof and posterior wall of the ordinary radical mastoid operation, when completed; in other words, at about the junction of the lateral with the sigmoid sinus, and passes into the sigmoid sinus.

In endeavoring to get as close to the jugular bulb as possible the surgeon should not press too far forward, as he is liable to injure the facial nerve, as it passes down through the posterior meatal wall on its way to the stylomastoid foramen. Bleeding may easily be stopped by carefully applied gauze rolls. In case free hemorrhage does not proceed from the bulbar direction of the sinus, a very serious condition confronts the operator, for, the blood current of the inferior petrosal sinus entering the anterior portion of the jugular bulb, on its way from the cavernous sinus, constantly playing on the septic end of the thrombus, naturally carries the infectious microorganisms down into the bulb, internal jugular vein, etc., toward the heart, lungs, liver, etc., thus readily producing sepsis, pyemia, metastatic abscess, etc., which usually result fatally.

It is, therefore, necessary that this end of a thrombus, or a thrombus located in this portion of the sinus, should be as thoroughly removed as possible, in order that the further or general spread of infection may be stopped. This may probably be best accomplished by the curette, which should be gently manipulated until free hemorrhage from below occurs,

which may be controlled by the rolls of gauze, bandage, etc., as previously described. It must not be forgotten, however, that it is usually quite impossible to successfully curette the jugular bulb by simply proceeding downward by way of the sinus. The curette may reach practically as far as the bulb and is then stopped by an intervening wall, an abrupt curve, etc., so that if a complete thrombus extends downward as far as the bulb or further, free hemorrhage will not usually be produced by sinus curettement.

Under these circumstances the operation of Grunert, Voss, Iwanoff and others has been proposed for the thorough exposure of the bulb. Owing to the complications and great danger of producing facial paralysis, hazardous hemorrhage, etc., however, it has never become popular and it is generally thought better to expose, ligate and partially remove the internal jugular vein and then clean out the bulb. It must not be forgotten that this does not always prevent the spread of infection by other channels, and that the sudden stoppage of the venous circulation may force infective particles backward into the meningeal and cerebral veins. While the internal jugular vein is, of course, the main avenue through which infection spreads from the sigmoid sinus to the heart and other organs, nevertheless, even with this principal channel ligated, infection may proceed through the posterior condylar vein, the occipital sinus and the mastoid emissary vein, all of which communicate indirectly with the subclavian and innominate veins, irrespective of the internal jugular. It will be remembered that the posterior condylar vein passes through the posterior condylar foramen and connects the lowest level of the sigmoid sinus with the deep veins at the back of the neck.

LIGATION OF THE INTERNAL JUGULAR.

Some surgeons invariably operate on the internal jugular before proceeding to open the sinus; others only ligate when free bleeding cannot be established from the bulbar end of the sinus; others open and curette the sinus and then wait for a continuance or exaggeration of the symptoms, when the jugular operation is performed. Certainly but few, if any, surgeons would sympathize with the views of Horsley, who only ligates after metastases have occurred, for then it would almost surely be too late. Favorable reports of jugular ligation in sinus thrombosis need not necessarily be attributed to

the ligation, as, for instance, Körner reports 43 per cent of recoveries without ligation. Still it is probably true that in septic and pyemic cases sinus operations with jugular excision yield better results than when the jugular is not disturbed.

My method of procedure is as follows: The sinus is opened, the head always having been previously lowered, the upper portion is curetted if necessary. If hemorrhage occurs freely from below the operation is finished; if not, the lower end of the sinus is carefully curetted and if free hemorrhage occurs nothing further is done. If free hemorrhage does not occur, pressure pads and a bandage are quickly applied and the jugular is ligated and excised, the bandage then removed, the sinus, bulb, etc., curetted and cleansed and the entire operative field, both in the bone and neck, properly dressed and bandaged. The necessity for the operation is greatly emphasized if the cervical tissues just below the mastoid are swollen and painful, and if any hardness and pain can be detected along the course of the vein in the neck, and if general pyemic symptoms are present.

I am convinced that excision of the jugular is not an operation which should be undertaken hastily, as grave circulatory and other disturbances are liable to ensue, and other methods of relief should first be tried, unless the patient's condition is very urgent. The literature furnishes many instances where sudden optic neuritis, edema of the brain, with hemorrhages and softening, thrombosis of other cerebral sinuses, etc., have followed jugular ligation, and it should not be forgotten that a small and insufficient jugular on the other side, totally unable to assume double duty, may be present and precipitate most unfortunate results. As a deduction from these observations Eagleton urges that in cases of sinus thrombosis, with optic neuritis, it should be concluded that the return venous flow is already seriously obstructed and that great caution should be observed in adding to such conditions by ligation.

EXCISION RATHER THAN LIGATION.

In case it is concluded to operate on the jugular vein it is better to excise most of it rather than merely to apply a ligature, as, if the jugular operation is necessary at all, its septic or pyemic current should be eliminated as much as possible and its poisonous contents removed. Before operating on the jugular the shoulders should be elevated on a sandbag or something of the kind and the neck and head allowed to drop

down, the latter turned away from the field of operation in order to place the neck well on a stretch, so that the anatomy of the parts is well defined and the tissues easier to operate on.

If the patient is a male the beard should be shaved and the skin antiseptically cleaned, care being taken not to disturb and break up a possible clot by rough handling. In the case of a male where it is at all suspected that a jugular operation will have to be performed the beard should be shaved before the mastoid operation is commenced. The neck should now be placed on a stretch and the anterior border of the sternocleidomastoid muscle clearly located and an incision (Fig. 4) made along its edge extending from its sternal attachment to the mastoid tip.[3] Bleeding vessels should, of course, be compressed at all stages of the operation so that a clear view of the operative field may always be obtained. The skin and other tissues should be kept stretched apart by blunt retractors. After the skin and superficial fascia have been completely severed the platysma myoides muscle with its oblique fibers can be plainly seen and should be fully exposed and incised along the anterior border of the sternocleidomastoid muscle, which can now be distinctly seen, and which should be fully uncovered by holding back the overlying tissues with retractors (Fig. 5).

The external jugular vein runs down the neck in a line drawn from the angle of the lower maxilla to about the middle of the clavicle and lies between the platysma and the sternocleidomastoid muscle. It crosses the latter muscle at an oblique angle, but does not cross the anterior border of the muscle except at its upper third at the angle of the jaw, where it may be seen, twice ligated, cut between the ligatures and gently retracted from the operative field. If ligation of the internal jugular were the only object to be attained it would not be necessary to touch the external jugular, for the ligature would be applied much lower down, near the cricoid cartilage, but as the intention usually is to excise practically the entire vein, it may be advisable to get the external jugular out of the way. It should be preserved if possible, however, as it performs an important part in caring for the venous blood after the internal jugular is removed.

Retractors should now be placed around the anterior border

3. Crile and others sometimes cut down on the sternocleidomastoid muscle and expose the vein, by separating the muscular fibers.

of the sternocleidomastoid muscle, and the muscle pulled back in order to expose to view the carotid, jugular, etc. This object may be facilitated by tearing and pulling the tissues apart by blunt instruments or the fingers, as care should be exercised that vessels and nerves are, as far as possible, uninjured, a thing difficult to prevent if knives and scissors are employed. The exposure should be made throughout the entire length of the sternocleido muscle from its mastoid to its sternal attachments, for it will be the object of the operation to remove practically all of the vein, and it should, therefore, be completely exposed (Fig. 6).

The deep fascia of the neck will be seen on retracting the sternocleido muscle, and on opening this and throwing it back the common sheath of the jugular, carotid and pneumogastric nerve may be seen. The utmost care should, of course, be taken to avoid wounding these structures, and as far as possible all sharp instruments should be eschewed in performing the delicate dissection. The internal jugular vein, the common carotid artery and the pneumogastric nerve are all encased in a common sheath. At the superior carotid triangle (the usual center of operative procedures) or at a point about midway between the angle of the jaw and the clavicle, the vein lies to the outer side of the artery, with the nerve between and posterior to both (Fig. 8), resembling, as Deaver says, the ramrod to an old-fashioned double-barreled shotgun.

ANATOMIC RELATIONS.

It should be remembered that while the relative location of these different anatomic constituents differs slightly in various portions of the neck, the vein always lies to the outside of the group and can be distinguished by its bluish-white color, by the fact that it becomes full and prominent on expiration and almost collapsed on inspiration or by pressing on it in the lower portion of the neck, when it will expand with blood (unless thrombosed) and quickly disclose its identity. The common sheath of the vein, artery and nerve should now be opened and also the septum which separates the vein from the artery and nerve, and the vein carefully dissected out, practically throughout its entire extent. The artery is, of course, easy to identify, both from its color and its pulsations, but the nerve may, unless considerable care is taken, easily be raised and ligated with the vein.

All enlarged and diseased glands should be removed. After the vein has been thoroughly liberated from its surroundings, the lower ligature should be placed as far down in the neck as possible, and the upper one high up, as close to the mastoid process as possible, using either ten-day chromicized catgut, black silk or Pagenstecher linen, preferably the first, as there is practically no danger nowadays in the use of catgut prepared by modern methods. Two ligatures are applied at each extremity by means of an aneurysm needle and the vein cut between each pair of ligatures, being careful to include nothing but the vein in the ligatures.

In order to check the breaking off and floating of small thrombotic particles toward the heart, etc., which may easily occur from the necessary surgical manipulations of the operation, the lowest ligature should be placed around the internal jugular vein as early as possible and as soon as a low exposure can be made, after which the regular steps of the operation may be proceeded with. This lowest ligature can usually be best applied at a point just below the angle of the omohyoid muscle. The tributary veins should be ligated, and they may be enumerated from below upward as follows (Fig. 7):

The middle thyroid in the lower third of the neck (Fig. 7, L); the superior thyroid (Fig. 7, H), a little higher up; the lingual (Fig. 7, F), a little below the lower jaw; and, lastly, the common branch of the temporal and facial (Fig. 7, D) veins. Sometimes these veins are so small as not to require ligating, with the exception of the common branch of the temporal and facial veins, which always requires careful attention. In removing the internal jugular it should always be severed above the posterior belly of the digastric muscle (Fig. 7, A) and above the common trunk of the facial and temporal veins, but the ligation need not extend more than half or three-quarters of an inch higher than this junction, as no more cervical veins enter the jugular above this point.

All of the veins which may require ligation enter the internal jugular (practically) in its anterior portion which if borne in mind will simplify their finding. After all connections are severed the vein may be removed and the parts thoroughly irrigated and cleaned preparatory to closing the wound, remembering that the operation should be performed with all the speed that is compatible with correctness and safety, as such patients should not be kept under anesthesia any longer

than is necessary. Sometimes inflammatory adhesions will so firmly unite the vein to its surroundings that its release will necessitate the expenditure of too much time, to say nothing of the danger of rupturing the carotid artery. Under these circumstances the vein and its tributaries should be split open, cleaned, and the neck wound dressed without permanent sutures.

It is not advisable to undertake to irrigate the sinus and what is left of the jugular, either from below upward or the reverse, as infection can easily pass into the inferior petrosal sinus. It sometimes happens that even after the jugular has been resected the upper ligature removed, and the sinus thrombus removed by curettement, there is no return flow of blood from the bulb, which indicates a thrombus of the inferior petrosal sinus. This is an infrequent complication, as is also a similar condition of the superior petrosal sinus, and should be met by following up the sinus and curetting it. The sinus should be cleaned of all possible thrombotic material by the curette after the jugular ligation, but forced flushing should not be attempted.

It is a good plan after the jugular has been resected to remove the upper jugular ligature and give the inferior petrosal sinus an opportunity to send its blood down through the opened jugular vein, in hopes of flushing away thrombotic debris that might be present. The vein should then be resutured, or, as suggested by A. Knapp, "on the day following the operation thrombosis having occurred in the jugular vein, below the bulb, this vein should be slit up, so that we have access to the bulb from both directions, thus preventing a purulent disintegration of the thrombi in the contributory veins and sinuses." Knapp recommends this procedure, especially in the severest cases.

TREATMENT OF WOUND.

The wound should now be drawn together except at its upper and lower extremities, which should be left open for drainage. The lower opening need only be very small, just large enough to admit of a round rubber or cigarette drain, which should extend inside the wound for perhaps two or three inches, and which should be gradually shortened from day to day until it is withdrawn altogether (Fig. 9). The opening in the upper portion of the wound should be larger and should be either packed with iodoform gauze or drained with a round

rubber or cigarette drain to be likewise gradually removed until compact healing occurs. Many surgeons do not suture this large neck wound, but draw the parts together with adhesive strips, feeling that the patient should be given every chance for his life by an open wound and free drainage. This, of course, involves an enormous scar but, nevertheless, is justified in bad cases, and possibly always. The jugular operation having been completed, the surgeon should now return to the sinus, on which work had been temporarily suspended.

The compress should be removed from the bulbar end of the sinus and all possible clot removed by means of a curette, after which narrow iodoform gauze should be gently but firmly packed into the interior of the sinus as far as possible. Pressure should, of course, be retained on the torcular end of the sinus and the entire mastoid wound dressed as usual, endeavoring as much as possible to separate the sinus from the mastoid packings. The neck should be abundantly dressed with gauze and cotton and the mastoid, head and neck enveloped in a voluminous and firm bandage, which it is best not to disturb for at least three days. Ordinary dressings at intervals of two or three days should follow until complete healing occurs.

TREATMENT OF SHOCK.

Should symptoms of shock occur during the steps of the sinus or jugular operation relief may be obtained by injections of physiologic salt solution into a vein, the breast or abdominal tissues or the bowels. If an intravenous injection is made, from sixteen to eighteen ounces of the solution should be used at a temperature of about 106° F. If the injection is made into the tissues or bowels from one to two pints should be used at a temperature of about 112° F. At all sinus and jugular operations appliances for making these injections should always be ready for instant use. In case the patient is in a very grave condition it is a wise precaution to give an intravenous infusion of physiologic salt solution before beginning operative procedures. Such infusions are also valuable in cases of general infection to counteract and combat the potentiality of migrating microorganisms.

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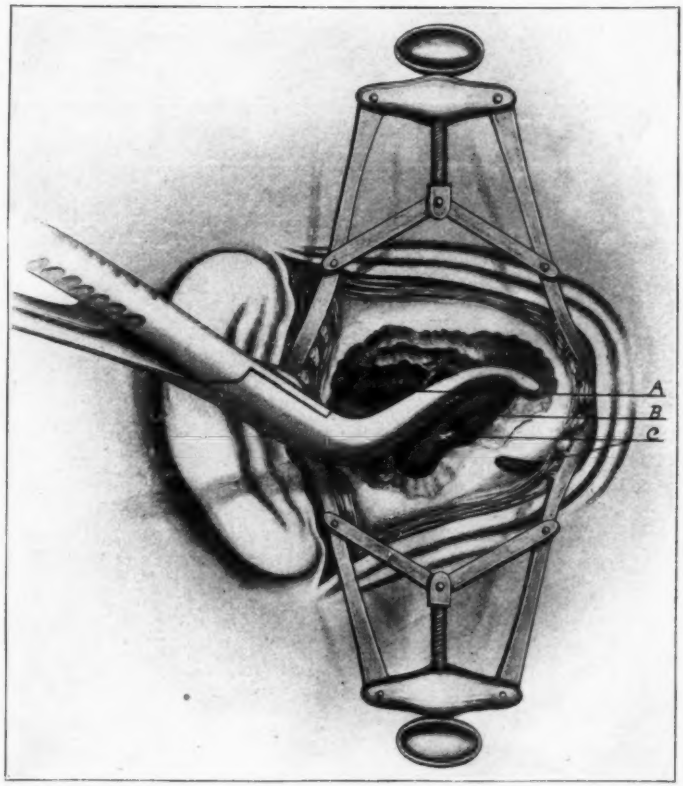

FIG. 1.

Exposing the sinus.
A—Mastoid antrum. B—Sigmoid sinus.
C—Mastoid emissary vein

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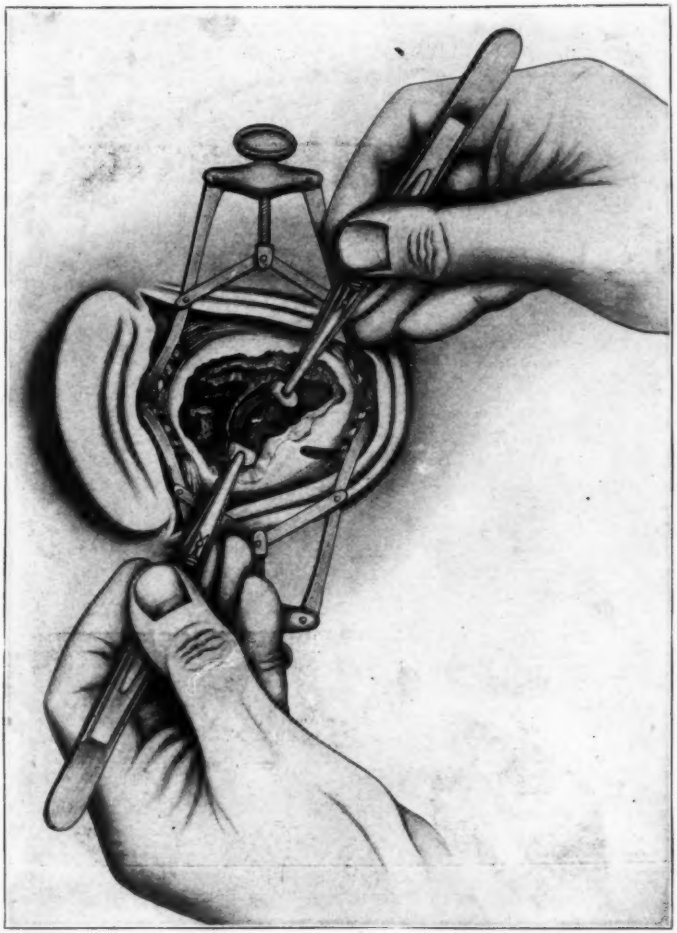

FIG. 2.

Compression of sinus to arrest hemorrhage, and to ascertain the presence of a thombosis.

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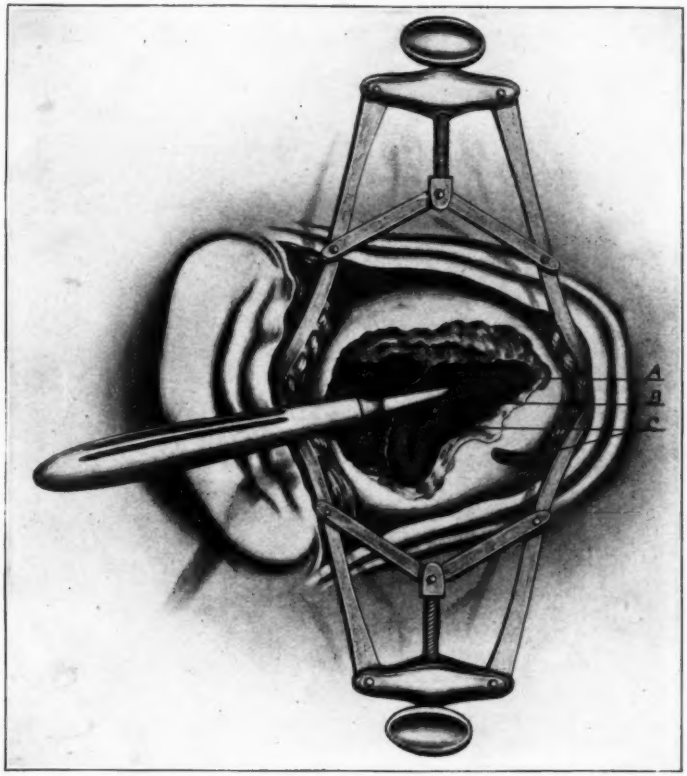

FIG. 3.

Curetting the sinus.

A—Mastoid antrum. B—Sigmoid sinus.
C—Mastoid emissary vein.

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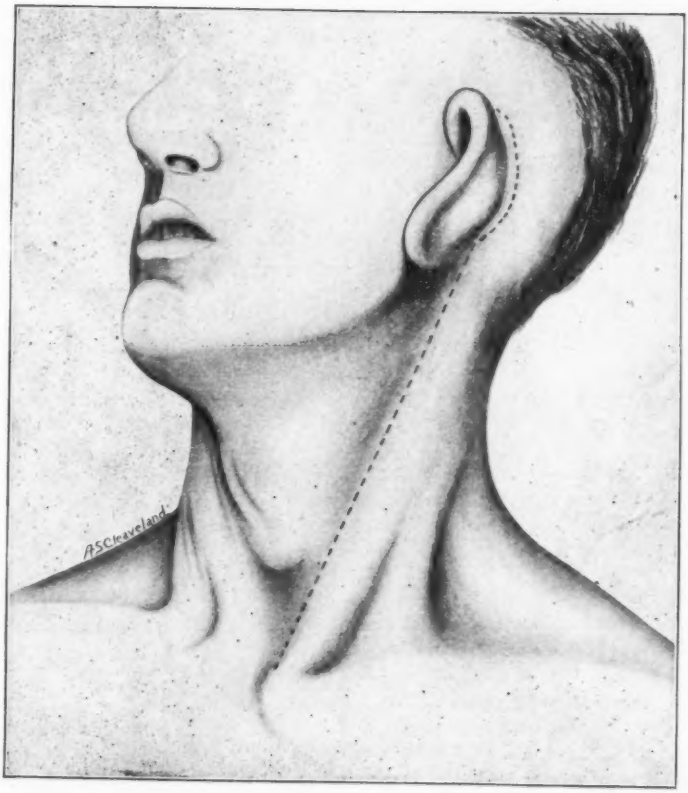

Fig. 4.—Initial incision for exposing internal jugular vein.

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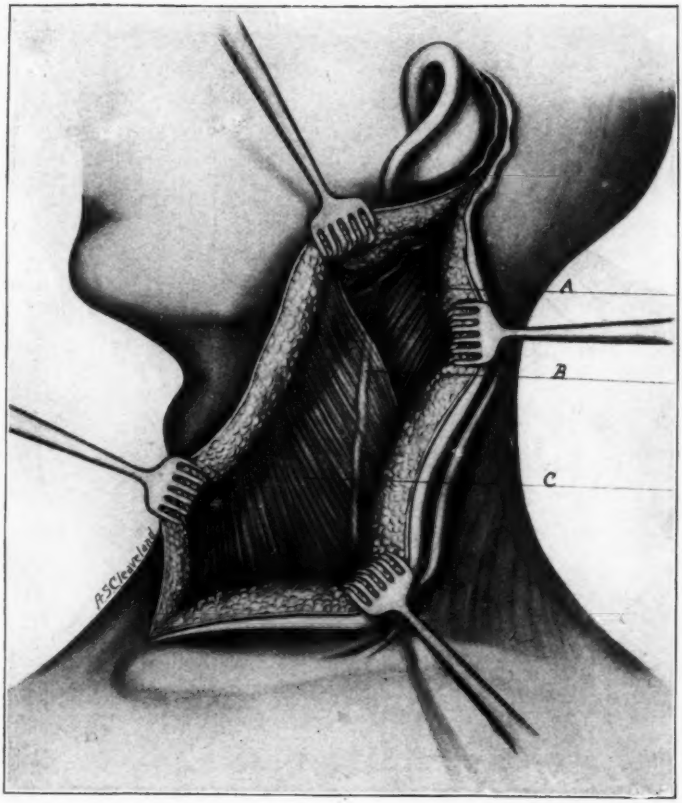

FIG. 5.

Exposure of the platysma myoides muscle and external jugular vein.
A—Sternomastoid muscle. B—External jugular vein.
C—Platysma myoides muscle

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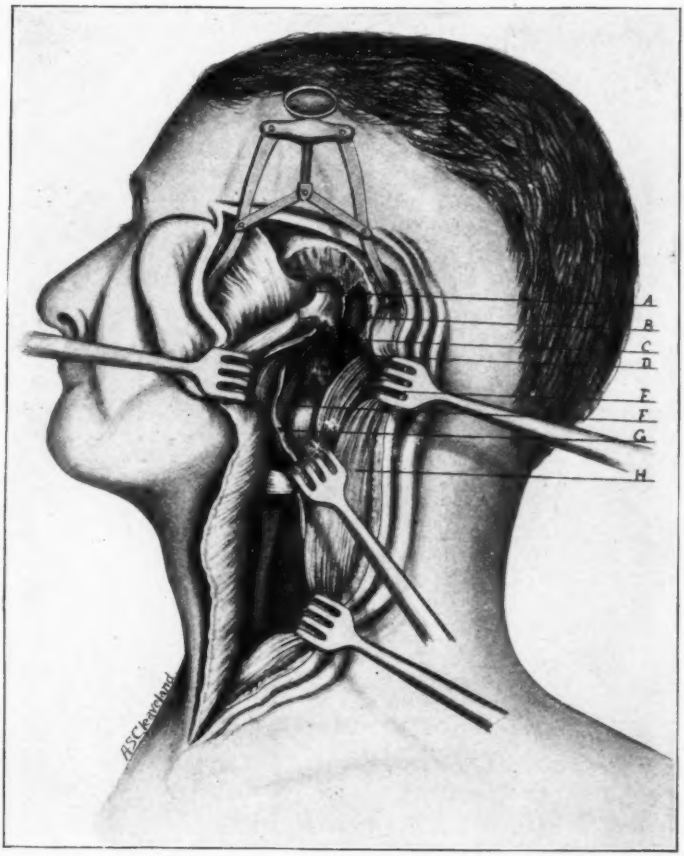

FIG. 6.

Demonstrating the course of the sigmoid sinus, jugnlar bulb and internal jugular vein.

A—External auditory meatus.
B—Sigmoid sinus.
C—Facial nerve.
D—Jugular bulb.
E—Digastric muscle.
F—Internal jugular vein.
G—Spinal accessory nerve.
H—Sternocleidomastoid muscle.

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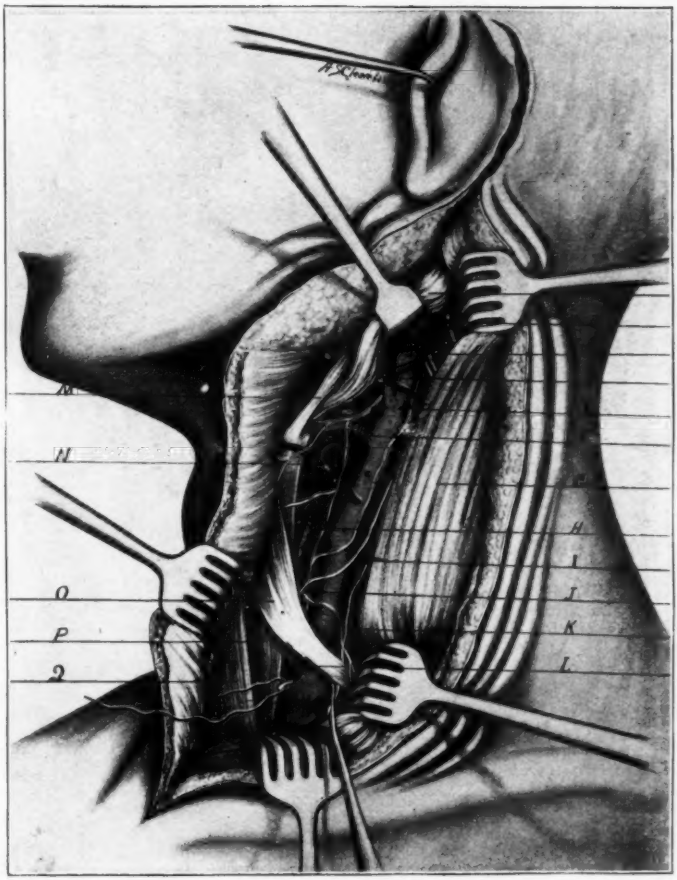

FIG. 7.

Exposure of the internal jugular vein, etc. The vein is about ready to be removed.

A—Posterior belly of digastric muscle.
B—Spinal accessory nerve.
C—Occipital artery,
D—Facial vein.
E—Hypoglossal nerve.
F—Lingual vein.
G—Sternocleidomastoid muscle.
H—Superior thyroid vein.
I—Communicantes hypoglossi nerves.
J—Common carotoid artery.
K—Phrenic nerve.
L—Middle thyroid vein.
M—Stylohyoid muscle.
N—Descendens hypoglossi nerve.
O—Anterior belly of omohyoid bone.
P—Sternothyroid muscle.
Q—Sternohyoid muscle.

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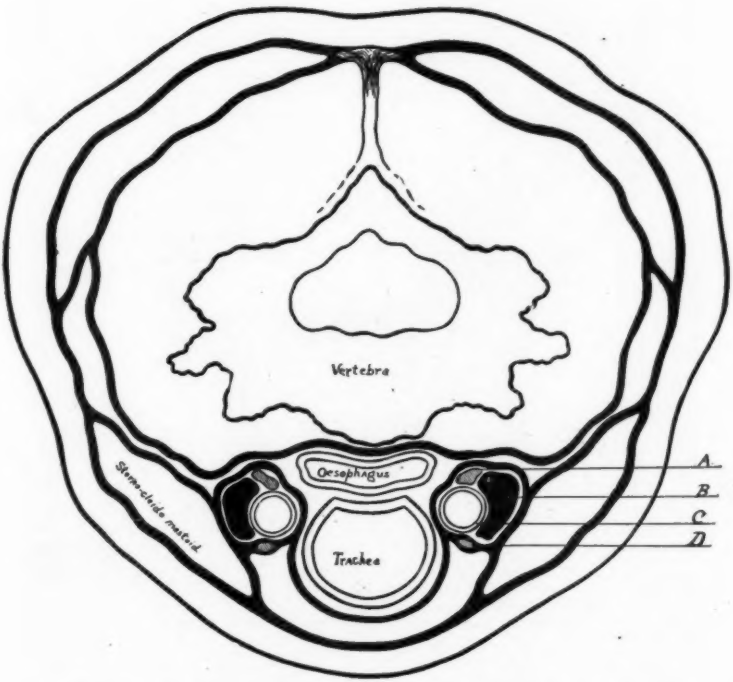

Fig. 8.—Cross section of the neck, after Deaver. A, pneumogastric nerve; B, internal jugular vein; C, common carotid; D, descendens hypoglossi nerve.

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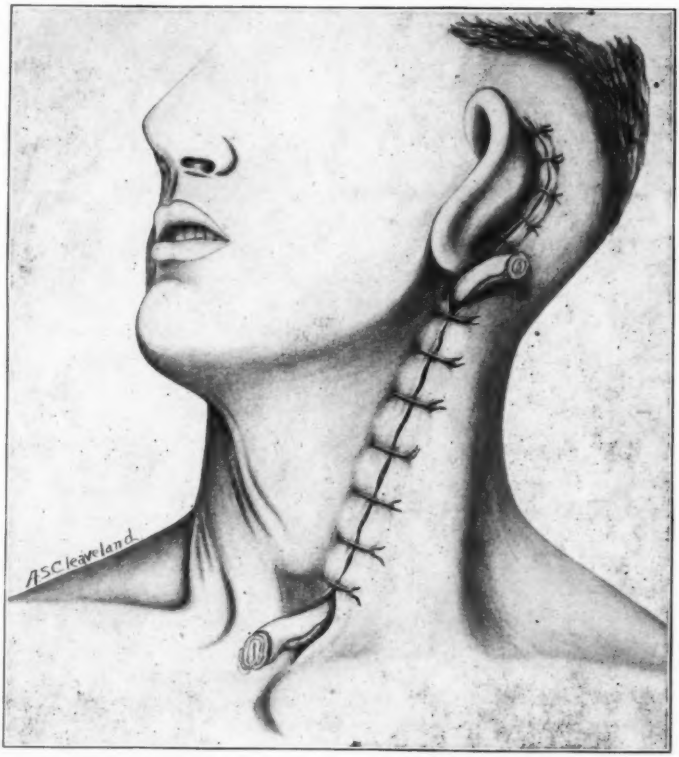

Fig. 9.—Closure of the wound, drainage, etc.

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XI.

TREATMENT OF THE INFECTIVE LABYRINTHITIS AFTER FIFTEEN YEARS' EXPERIENCE.*

By Dr. Albert Jansen,

Berlin.

When the title of my paper was forwarded I was unacquainted with last year's transactions of the American Laryngological, Rhinological and Otological Society, and consequently the excellent papers upon this subject, especially that of Dr. Richards, were unknown to me. Had it been otherwise, I should certainly have selected another subject, being fully aware that, from my observations, not many new or original facts can now be presented.

May the fact of my having proposed the labyrinth operation, and of having carried it out for fifteen years, serve as an excuse in your eyes for the choice of this subject.

My observations are based upon upwards of one hundred labyrinth operations. In only two cases was the cochlea alone operated upon. In twenty-three cases the cochlea and vestibule were opened together, and in the remaining cases only the vestibular apparatus. Only five of these cases followed acute middle ear suppuration.

Among those operated upon, 29 died, or 25 per cent, excluding four, in which death was not due to the labyrinth suppuration.

In sixteen cases the operation was performed after purulent meningitis already existed, and death could consequently not be prevented. In three others death ensued undoubtedly from brain complications, like cerebellar abscess, caused by the labyrinth infection.

I propose to arrange my subject synoptically into three or four groups.

The first group which, from its origin and course, is clearly distinctive from the remaining cases, comprises the infectious

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labyrinth affections caused by lesions of the labyrinth, principally at the oval window.

In this first group of cases we are fortunately able to observe the origin of the disease. Therefore, the symptoms observed assume a decidedly diagnostic importance.

Injuries to the labyrinth are frequently caused by the radical operation. The horizontal semicircular canal is the part most liable to injury; this injury is well known and comparatively frequent.

But of this kind of lesion I shall not speak here. I will only mention that it is generally recognizable at the moment of origin. It seldom leads to operative opening of the labyrinth or to fatal consequences.

In any case it is unnecessary to perform at once an opening of the entire vestibular portion on account of this lesion. Certainly the necessity may arise during the process of healing, but it is evidently exceedingly rare, and I can recollect only one case where the opening of the vestibular apparatus was afterwards necessitated.

We shall here treat of the lesions of the labyrinth, which are much more dangerous in their consequences; namely, the lesions of the fenestra ovalis and the luxation of the stapes.

This part of the middle ear is the one most exposed to lesion from curetting, and, excepting the horizontal canal, the part mostly endangered at the radical operation. Sometimes the luxation, in the case of stapes disease, is very easily produced. Through the accidental removal of the stapes, the vestibule is exposed to serious danger, but the accident is more readily recognized and the danger thereby diminshed.

As a rule, however, it is only a question of luxation of the stapes, of a partial rent of the ligamentum annulare, of an insignificant injury which, in opposition to the injured horizontal semicircular canal, and to the removal of the stapes, we do not notice at the moment it occurs, and which we perceive afterwards only from the symptoms.

That we are dealing with a luxation of the stapes is at first merely a supposition. Experience teaches us, however, that this supposition generally proves correct. The great danger of the stapes luxation lies in the fact that under necrosis it occurs unnoticed and that infection of the vestibule immediately results.

In its consequences, as a rule, in the beginning, such an in-

jury is far more disastrous than the opening of the semicircular canal, while the initial clinical symptoms are usually far less alarming.

Half a day or more elapses before the first signs of labyrinth infection appear, and generally still more time is lost before they are properly appreciated. When the accident occurs, let us say, from curetting in the after-treatment of radical operations, we can make the following observations: Lesion of the labyrinth at once manifests itself in vertigo, disturbances of equilibrium and nystagmus.

It is true that these labyrinthine symptoms sometimes assert themselves only for a moment, coming like a flash of lightning and as quickly vanishing, often so suddenly that the nystagmus is no longer demonstrable.

Directly after the lesion, owing to the appearance of vertigo, the head jerks backwards or forwards, away from or towards the doctor. Sometimes both the head and the upper part of the body make this jerk. Not infrequently there is nausea or vomiting. For a few moments the patient is seized with violent vertigo; he is afraid of falling off the chair, everything seems to be turning round him or with him. On rising, he seems unsteady on his feet, totters or falls.

Occasionally a subjective sensitiveness of the auditory nerve is present in the form of ringing or buzzing. On examining quickly for nystagmus, it is usually found to be present, sometimes with quick, short oscillating movements rather than with slow and long movements, and always strongest when the eyes are directed to the healthy side, or only in this direction. Vertigo appears sometimes only for a moment; sometimes it persists for several minutes, sometimes for a few hours.

Frequently, on account of the overpowering influence of pain, the patient is not conscious of vertigo when it is of short duration, and does not complain of it. The physician mistakes these jerky movements caused by vertigo for a reaction from pain. Even after a few minutes the patient can often rise from his seat and move about again quite well. The nystagmus often disappears, too, and only returns as soon as the infection is established, but continues in case of labyrinthine hemorrhage. It is very easy to overlook the lesion with such feeble labyrinth symptoms. And now, when we have an out-patient to treat, we are confronted by a disastrous chain of circumstances.

The dressing of the wound is done in the usual way, and as the scraping out is often followed by hemorrhage, the packing is done tighter than usual, for hemostatic purposes. This is another unfavorable moment, and facilitates the infection of the vestibule. The patient returns home; soon he feels worse, and goes to bed. The following day he does not feel able to go out; he stays in bed, the dressing remains. The infection progresses and the case ends fatally.

A much more favorable view may be taken of the patient's fate if the damage is perceived at once, or if the patient is undergoing clinical treatment, and consequently, an early ascertainment of the labyrinthine lesion is possible. The damage being at once perceived, a great deal may be done to render the complications less destructive in their course.

One must avoid any manipulation which is not absolutely necessary; powder with iodoform, push in loosely a piece of gauze for absorption of secretions, and attend to the constant discharge of secretion by frequent change of dressings. With extreme care and absolute rest in bed, labyrinth infection is not infrequently prevented.

My cases of labyrinth injuries are 19 in number.

1. Accidental extraction of the stapes..... 1
2. Luxation of the stapes (the diseased one) during dressing..... 1
3. Luxation of the stapes (the diseased one) during curetting..... 7
4. Luxation of the stapes (the diseased one) during the radical operation..... 6
5. Fissure through the horizontal canal..... 1
6. Lesions by galvano-cauterization*..... 3

19

7. Infection by the curetting of a labyrinth previously exposed by operation..... 4

Spontaneous cure occurred in two cases; in the one, after scraping out, in the other one after accidental extraction of the stapes. In the latter case the symptoms from the labyrinth lasted several days; they soon diminished and at last disappeared entirely with good hearing.

The second was affected with severe labyrinthine symptoms after scraping out, and subsequently for some weeks with

*These cases showed a slow course.

severe symptoms of meningitis. At examination a good many years later the ear was deaf without caloric reaction. There was no disturbance of equilibrium.

Thirteen were operated upon with four fatalities, 37.7 per cent.

Life was saved:

1. In 3 cases with the complication of serous meningitis.

2. On account of the severe extension of the labyrinth disease in two cases.

It is interesting to know how long after the infection death occurred.

I. In the non-operated cases:

In 2 cases after 7 days.
In 1 case after 11 days.
In 1 case after 18 days (empyema of the saccus endolymphaticus).
In 1 case after 37 days (galvano-cauterization).

II. In the cases operated upon:

In 2 cases after 6 days.
In 1 case after 9 days.
In 1 case after 11 days.

III. In those cases which were curetted after opening the vestibule:

In 1 case after 5 days.
In 1 case after 11 days.
In 1 case after 27 days.

In half of all cases death occurred in from 5 to 8 days.

Further, it is interesting to note on which day after infection the operation was performed.

I. In the cases resulting in recovery:

1 after 24 hours.
1 after 27 hours.
1 on the fourth day.
1 on the sixth day.
1 on the eighth day.

In two cases two months after operation and in the cases arising from galvano-cauterization:

1 case the thirteenth day after infection.
1 case the fifteenth day after infection.

II. In the cases resulting in death:

The second day after lesion, 4 days before death.
The second day after lesion, 4 days before death.

The third day after lesion, 6 days before death.
The fifth day after lesion, 6 days before death.
The tenth day after lesion, 1 day before death.

We see that in two cases the operation was performed too late, even on the second day after lesion; also in another case, on the third day after the lesion, whilst still another case resulted in recovery, though the operation was performed four days after lesion, and recovery has even taken place, eight days after lesion, as well as two months after. The longest period intervening between the operation and death was six days.

The question remaining for discussion is, whether in these cases of injury to the labyrinth death was accelerated or occasioned by the operation.

In the cases presented early for postmortem examination or for operation, the tissues within the labyrinth always showed a hemorrhagic appearance.

The membranous labyrinth was deeply stained with blood, sometimes blackish red, always swollen, sometimes considerably so; the earlier seen the more hemorrhagic it was. As a rule, the process was somewhat more advanced in the vestibule, sometimes when older it showed a greenish discoloration, occasionally also of the bone. The contents of the membranous canals were mostly light red, occasionally dark red.

In one case, indeed, there was after 27 hours, a chocolate-colored mass in the horizontal semicircular canal, the vestibule showing a fresher red. In the early cases no pus whatever was found at operation or postmortem. In one case of postmortem examination after 18 days, the vestibule and cochlea were found filled with pus, also the much dilated saccus endolymphaticus. In cases that came under operation or postmortem later, great destruction was evident in the labyrinth.

These cases, if not operated upon, would probably have met the same fatal end. There is nearly the same period between death and lesion in the operated and non-operated cases. Thus we cannot conclude that the death was accelerated or occasioned through the labyrinth operation.

Some of these lesions were rapidly followed by a very infective purulent meningitis. One patient showed the characteristic symptoms of meningitis two days after the injury to the labyrinth.

As only two out of the 19 cases resulted in spontaneous cure, that is, 10.5 per cent, and even 1 of 17 with luxation, that is,

5.5 per cent, we come to the conclusion that in the case of a determined labyrinthine infection it is better to open the infected part than to wait long for the labyrinth symptoms to increase or diminish.

But how are we to recognize labyrinthine infection? By the rapid increase of the labyrinthine symptoms, often in connection with the signs of inflammation: headache, coated tongue, fever, reactive inflammation of the meninges.

According to pathologic facts the question may arise whether there exists:

1. The purely traumatic injury only.
2. Simple irritation with or without injury, with or without hemorrhage (labyrinthitis serosa hemorrhagica).
3. Infectious labyrinthitis.

In the first two conditions there is a good chance of spontaneous cure, while in the third there is a great tendency to go on to fatal ending. In the first group, inflammation is wanting; in the second group, inflammation is slight; in the third group, inflammation and infection are severe.

In most cases we have an injury of the vestibular apparatus, and in consequence symptoms of vestibular irritation; that is, first of all, dizziness, disturbances of equilibrium, nystagmus. If these symptoms are produced, for instance, by pressure upon the loosened or luxated stapes, the loosening of the dressing is sufficient to remove them. But if they continue, and even increase, they are proof of a severe and growing disturbance in the labyrinth that cannot be explained by the luxation of the stapes alone.

Sometimes the hemorrhage gives the explanation, but that is seldom the case. The hemorrhage may be caused by injury as well as by infection. If now pulse and temperature increase equally, the situation becomes clearer, but at the same time more dangerous. The higher temperature is hardly produced from the infected vestibule or labyrinth, but in most cases (together with the pulse augmentation) by the reaction of the arachnoides. As soon as the temperature increases, be it perhaps only to 38 degrees or so, the indication to open the labyrinth is absolutely clear; but it would be wrong, in case of no fever, to neglect the other symptoms. We have seen that in one case the malady continued for 8 or 10 days without fever, the latter only setting in 12 hours before death.

In cases without fever also it seems to me more desirable

to perform the operation than to leave it, as soon as the symptoms of labyrinthine infection are marked, persistent and on the increase, because it is much more probable that the malady will develop into an extensive labyrinthine affection, which would demand an operation later, perhaps under less favorable circumstances. Even an exceptional spontaneous cure cannot turn me from this opinion.

How are the labyrinthine symptoms represented? There are nystagmus, vertigo, disturbed equilibrium, as symptoms of vestibular affection; the loss of hearing is the sign of the cochlear affection. In cases of lesions of the oval window all these symptoms of labyrinthine disease must be searched for in the early stage; they are not so evident as to obtrude themselves upon our notice. If the patient remains in bed after the operation these disturbances are not apparent. Nystagmus is slight but distinct. On looking straight in front the eye seems to rest quietly. On examining sharply with a bright light we observe, when the eyes are directed straight in front, a fine, slight, rapid rotary nystagmus, oscillating towards the healthy side. As soon as the eye passes the middle line towards the healthy side, the twitchings increase, moving in long jerks, and being either rotary or horizontal. The nearer the end-point the more extended the twitchings. They are often now so numerous that they cannot be counted, mostly remaining just about countable. In five seconds one may count five, ten or twelve oscillations, which are not regular and which sometimes cease.

In slight cases the nystagmus ceases if the eyes are directed toward the affected side. In severe cases it continues; and in other less severe cases it reappears, but then oscillating toward the affected side and more slowly. The growing intensity of the twitchings is a symptom of the progress of the inflammation in the vestibular apparatus.

Lying in bed, dizziness is scarcely or not at all observed. The patient lies with closed eyes, his head pressed into the cushions, without moving. He avoids movements, which might produce dizziness, and so the patient, not being aware of the sense of dizziness, cannot complain of it. As soon as he is made to turn upon the injured side or to sit up, he will feel dizziness. Should the patient be requested to get up, vertigo would be more pronounced. When the patient is in bed disturbance of the equilibrium does not appear. If free sitting

up in bed is impossible without effort and wavering, the disease must be a very severe one. In the early stage the patient is often able to walk without any unsteadiness. The patient himself may not have the slightest sense of disturbed equilibrium, not even when walking with closed eyes. If asked to place one foot before the other, there comes a slight wavering, sometimes only when the eyes are closed. With this experiment of standing with one foot before the other and the eyes closed, one is able to prove any degree of disturbance of equilibrium which otherwise is not observed.

The calor'c reaction, which serves us so well in the diagnosis of labyrinthine disease and gives us very exact knowledge of the degree of sensitiveness in the vestibularis, is really of no use here. In these cases where we must presume that there is an artificial passage, made by injury, leading to the labyrinth, we have to avoid syringing as long as we may hope to get a spontaneous cure. But putting in cotton with cold or hot water instead of syringing does not give reliable results. Moreover, we are observing the labyrinthine inflammation from its beginning, when there is no weakness of the vestibularis. The inflammation may rapidly progress toward the brain before the loss of the vestibular function. We should not forget to enumerate here headache and coated tongue as signs of an inflammatory process in the vestibule, although they may be produced by meningitis as well as fever. We do not doubt that in all these cases of labyrinthine injury in the early stage the vestibularis will very well react to cold and heat.

In this early stage we are not often able to diagnosticate an infective meningitis. The symptoms are still deficient. By the lumbar puncture we may in the earliest stage find a minimal augmentation of pressure, which, however, may quickly rise higher; a few times we found diplococci in clear fluid, once even after 24 hours; a little later we find more leucocytes with it, and a turbid fluid. Among my cases, however, there are some of purulent meningitis without any considerable pressure augmentation, with no bacteria in the lumbar fluid, and even a clear fluid. That is why the negative result of the lumbar puncture can scarcely be used as a contraindication against the operation. The operation ought to be done as soon as possible, in order to prevent the development of an infective purulent meningitis.

The following clinical picture is typical for a severe and progressive infection of the labyrinth:

The patient lies apathetically in bed, avoids moving his head or even raising himself in bed, refuses food and drink because of the inclination to vomit. He lies quietly with closed eyes, mostly on the healthy side; from time to time vomiting occurs, especially after taking nourishment or after moving. The tongue begins to get coated. Slight headache appears. On attempting an upright posture or on turning around he becomes dizzy. Sitting in bed is difficult or impossible. He gives the impression of being very ill. Nystagmus is manifest in the above-mentioned way, at first slight twitchings, increasing to quicker, more lengthened and irregular twitchings, rotary and horizontal oscillations, ten to twenty and more in five seconds, scarcely countable. They are very strong in the end position to the healthy side, continue looking to the affected side.

It is only when the patient gets up that disturbances of equilibrium are observed; at first by standing with feet placed one before the other, in severe cases with open eyes, or standing with feet together and eyes closed. At this stage I consider an operation indicated, although there may be no increase in temperature. In case of the slightest increase in temperature, if this is not due to the general wound conditions, the operation is imperative and must be performed immediately.

We have seen that in the opening of the vestibule the operation was performed too late even two days after the lesion. If the infection has penetrated from the vestibule to the arachnoid it is uncertain whether we shall be able to check it. Unfortunately, we have often a streptococcic infection. On the other hand, if the infection has not yet penetrated the nerve canals, we may be able to arrest the process by a wide opening of the vestibule.

Symptoms of the cochlea are inconstant. Sometimes humming or ringing is experienced, frequently nothing.

The arachnoid membrane is exceedingly early affected by the inflammatory processes of the vestibule. In two cases lumbar puncture proved the existence of pneumococci in the fluid 24 hours and 27 hours after infection. The pressure had risen only very slightly to 150 mm. In this early stage leucocytes are not yet present. In another case lumbar puncture showed a pressure of 220-300 mm.; it was transparent, free from bacteria and leucocytes. Once it was found transparent after 48 hours, and had only a pressure rising to 150 mm. In one case the liquid was clear even after four days, with a pressure ris-

ing to 290 mm. In a fifth patient, however, the cerebrospinal fluid was turbid, but free from bacteria, with a pressure of 220 mm.

Just in the first few days when we are looking for symptoms to fix the diagnosis and to decide the necessity for operation, the lumbar puncture seems to give us rather unreliable results, though in spite of this it ought to be done.

Fortunately, not all cases end so tragically, in which, directly after the operation, we notice those disquieting symptoms indicating labyrinthine affection. In my cases I have been able to include twelve with important labyrinthine symptoms arising directly after the operation or a few days later, dizziness, vomiting, nystagmus, disturbed equilibrium.

All these cases were cured promptly.

Moreover, in a number of cases—eleven or more—labyrinthine symptoms appeared during the first week after the operation.

With these patients the symptoms of labyrinthine irritation were of long duration, for weeks and even months.

Two possibilities are here presented. Either in all cases, or in a part of them, there is a luxation of the stapes or an infection of the vestibule induced by the injured cochlea, or there is a latent labyrinthitis, aggravated and made manifest by the operation or by the irritation accompanying the wound healing.

At the same time these cases prove that symptoms of labyrinthine disease do not give us a right immediately to operate in every case, or to oblige us to open the vestibular portion, before satisfying ourselves of the infectious and progressive character of the complication.

My numerous observations of the labyrinthine lesions have taught me that the greatest danger is imminent from the infectious disease of the vestibular portion and essentially from the vestibule itself. After having laid bare the vestibule we may generally consider the danger obviated. We usually combine the exposure of the vestibule with that of the ampullae. But we do not pay any attention to the semicircular canals, from which we fear no danger. There are no free pathways by which the infection from the semicircular canals could get into the inner portion of the cranium. The smaller a canal is the sooner it is cured of inflammatory processes.

Although there exist some free channels leading from the

cochlea and capable of conveying infection into the inner portion of the cranium, we have to deal with narrow canals similar to those of the semicircular canals, and the danger becomes greater only when, by bony destruction, the narrow canals form a larger cavity with or without sequestra.

This seldom or never happens in cases of a rapid course. The infection arises in the vestibule, the disease extends into the vestibular apparatus, the symptoms are those of vestibular irritation, and if we interfere as soon as the welfare of the patient demands, a severe, infectious process can scarcely have had time to develop in the cochlea.

These theoretical considerations agree with the experience we have had in such cases, and the results which I have already stated.

The circumstances change when the infection is produced in the cochlea, or when the cochlear irritations have a share in the form of the disease known as humming or ringing in the ear, or noises of various kinds, or in processes having a slow course, where the development of the disease took many weeks so that severe affection of the cochlea may have taken place. Then the exposure of the cochlea may be indicated.

In the literature we find numerous proofs of this complication, as in the pages of Hinsberg, Freytag, Friedrich, Heine, Zeroni, Alexander, but I have noticed in these reports that rather too often the labyrinth infection is interpreted as a latent labyrinthitis, aggravated by the radical operation.

Operations on the Labyrinth Resulting in Recovery.—I come now to the second group. There are 62 patients. The vestibule was opened 63 times, the cochlea 11 times; in one case the cochlea alone was opened.

Excepting two cases the disease was always chronic middle ear suppuration which had caused labyrinth infection. In the two acute cases a deep extradural abscess penetrated into the labyrinth at the posterior petrous plane.

Occasionally the affection was latent. The diagnosis was made either before operation, as was generally the case, or during the operation, through the evidence of fistulae in the labyrinth wall or in the oval window, which was then usually found to be open. Occasionally the labyrinthine disease appears quite spontaneously during treatment, a long time after the mastoid operation.

In the cases which, before the operation, had shown severe

labyrinth symptoms, these were, as a rule, greatly relieved after operation; often, on awakening from the anesthetic, the patients felt free from vertigo and much easier in the head; they could sit up in bed and move much better.

Inversely, those cases which presented slight labyrinthine symptoms frequently exhibited after the operation either slight or much stronger symptoms of nystagmus and vertigo for a short time.

In cases which, before operation, were free from vertigo and labyrinth symptoms, these latter occurred after the operation, now and then in a highly distressing degree.

Moreover, these appearances have been frequently met with by other authors. With striking frequency we read in the history of patients of the great increase in labyrinthine symptoms after the opening of the labyrinth. These appearances were manifest very strongly in one case immediately following the radical operation, when the fenestra ovalis was found open; there was a strong suspicion of traumatic affection, recently caused or increased by probing. The condition found at the operation, however, contraindicated this, since the membranous labyrinth was glazed, swollen, not hemorrhagic.

In some few cases the labyrinth operation produced no changes whatever in the health of the patient; once I even found a child sitting up in bed and playing four or five hours after operation.

In one case violent vomiting occurred at the moment of opening the horizontal semicircular canal, and still another case experienced the sensation of being suspended in the air at the moment of striking off the lower vertical semicircular canal, where the vestibule was opened. The operation was performed without an anesthetic.

In one case a discharge of cerebrospinal fluid came from the vestibule. On the other hand, we never observed any fluid discharge among this series of cases upon the opening of the cochlea; quite in contrast to the cases which ended fatally.

As complications we sometimes find sinus thrombosis without any connection with the labyrinth affection; once, cerebellar abscess twice, purulent meningitis; in a number of cases, serous meningitis.

Serous meningitis is a common complication in infective labyrinth disease. It is principally localized in the posterior cranial fossa, while the middle and anterior fossae are free

from it. In the puncture of the dura we found cerebrospinal fluid in great quantities only in the cerebellum.

Several times I could find proofs of an interesting affection of the saccus endolymphaticus.

In several cases I found a circumscribed livid discoloration, about the size of a five-cent piece, on the cerebellar dura, from which the surrounding dura stood out rather sharply, owing to deficiency in livid discoloration and to its less moistened appearance. This area was close to the descending crus of the sigmoid sinus. I have often observed this phenomenon. There is no doubt, in my opinion, that it is a sign of an inflammatory process of the saccus endolymphaticus. The appearance depicted by this circumscribed livid discoloration in the vicinity of the sinus is a very characteristic one, and since recognizing this phenomenon and searching for it I have come across it repeatedly. It is a serous inflammation of the saccus which is in some cases productive of purulent inflammation.

When the radical operation has been performed, the closest inspection of the labyrinthine capsule is necessary.

In 1896 I reported 196 cases of defects of the semicircular canal, which, with few exceptions, were located on the horizontal semicircular canal. Since then I have seen 350 cases of infective labyrinthine disease, of which again by far the majority were fistulae of the horizontal semicircular canal.

Although fistula of the horizontal semicircular canal is much the commonest, fistula in the fenestra ovalis is by far the most dangerous. Exploration of the fenestra ovalis has only been systematically carried out by me of late years. Among the 62 cases resulting in recovery, the fenestra ovalis was found empty in 20 cases, and the horizontal semicircular canal was mentioned as intact in 10 cases; in several cases with transparent lumen.

The presence of fistula in the horizontal semicircular canal is doubtless the chief pathway for the conveyance of infection to the labyrinth; but certainly not infrequently it must also be regarded as an escape from within.

When the invasion of the labyrinth occurs through the horizontal semicircular canal, there is present in many cases a circumscribed labyrinthine disease, the affection being limited to the semicircular canal.

It is by no means always easy to arrive at a correct conclusion respecting the condition of the fenestra ovalis. The

fenestra ovalis must be quite exposed for examination which must be conducted with great precision and delicacy, lest we ourselves cause a lesion in or at the window. We must also bear in mind that infection of the vestibule, if the vestibular wall is intact, may arise from the cochlea, which may be infected by an inconspicuous fistula in the promontory, or again, through a diseased fenestra rotunda.

It is now important to decide whether we are dealing with a circumscribed disease of the semicircular canal, with a diffuse disease of the vestibular apparatus, or with a disease of the entire labyrinth.

Unquestionable cases have come under my notice in which the entire vestibular portion was attacked, but where the inflammation was restricted, for instance, to the horizontal semicircular canal. The entire labyrinth can be attacked by a non-infectious inflammation.

The part attacked is, without doubt, either impaired in its functions or completely paralyzed. The cochlea would appear to become paralyzed in its functions more quickly than the vestibularis. The cochlearis is the more highly organized nerve, and the higher the organization, the more delicate it is, and consequently, the greater the liability to decay and loss of function.

The following kinds of labyrinth affection may occur:

I. A serous, non-infectious inflammation of the entire labyrinth, or of separate parts of it, produced by the adjacent middle ear disease, with or without defect in the labyrinth capsule.

II. An infectious disease of separate parts of the labyrinth: (1) the cochlea; (2) the vestibule; (3) the semicircular canal. There may be:

(a) A serous hemorrhagic infection.
(b) A purulent hemorrhagic infection.
(c) A purulent infection.
(d) Infection with necrosis.

The partially infective inflammation may be associated with a diffuse non-infective reaction of the entire labyrinth.

In every case of inflammation of the cochlea the function quickly ceases. If there is a serous affection or a quite slight infectious one, the process may result in recovery with a complete or, more probably, a partial restoration of function.

In a serous or slightly infective disease of the vestibular apparatus, the functional powers appear able to exist for a longer

time. Here recovery may also be expected with complete restoration of the function. If the patient's hearing is fairly good, we have, most probably, a circumscribed disease of the semicircular canal.

More important and significant for the diagnosis are the phenomena of vestibular disease, signs of irritation or paralysis of the nerve. In the manifest cases the diagnosis may often be found at first sight.

Barany's method of caloric reaction most promptly acquaints us with the condition of the vestibularis and its functional power. We herewith test its sensitiveness by the application of cold and heat, by temperatures above or below blood heat. We use cold water of 12 degrees to 15 degrees C., and hot water (50 degrees). With susceptible nerves slight differences suffice, with less susceptible nerves great differences in temperature must be applied. The results so obtained are especially valuable in those cases where the labyrinth symptoms have already disappeared, where the labyrinth disease is latent, or where it has resulted in spontaneous cure with paralysis of the nerve.

In syringing with cold and hot water, characteristic nystagmus and disturbed equilibrium combined with vertigo are produced. Nystagmus appears on the application of cold exactly as in the case of labyrinthine suppuration, on the opposite side, and, vice versa, on the application of heat, it appears on the same side, its intensity depending upon the temperature or the pathologic condition of the nerve. We observe here all degrees of nystagmic movements from quite slight, scarcely perceptible, twitchings, to rapid, wide, wild and irregular rolling of the eyes. Similar to this are the disturbances of equilibrium, which sometimes do not exist at all, sometimes only appear in slight degrees, and sometimes in so high a degree that the patient has no longer any control over himself. He is able neither to sit upright nor to stand, and in these severe cases the vertigo produced by rotary movement is extremely pronounced. Everything seems to be turning round him and he himself collapses and falls. In slight degrees vertigo is sometimes completely absent.

The appearances produced by caloric reaction are perfectly in keeping with the phenomena of manifest labyrinthine disease.

Though it was known long ago that syringing with cold

water produced vertigo, great merit is due to Barany for having followed up these symptoms most thoroughly, and for having constructed a method of practical use with the patient, which in latent cases of labyrinth disease enables us to arrive at a prompt conclusion of the condition of the vestibularis; but not of the kind of pathologic alterations.

If no reaction occurs, paralysis of the vestibularis is proved. In most cases a purulent or infectious process in the vestibular apparatus will be the cause.

To answer the question concerning indications for operation, the caloric reaction has to be used with precaution, and in those cases of very rapid development of labyrinthine inflammation, which are so very dangerous because of the possibility of a rapid infection of the arachnoid, it is also unsatisfactory. In these cases the caloric reaction has not yet disappeared at a time when an operation would give the best chance. In those cases where the operation allowed me to ascertain the condition of the inner labyrinth, caloric reaction was as follows: Four times negative, twice almost negative, neither vertigo nor nystagmus, but immediately after the test a severe fainting fit occurred, and the spontaneous nystagmus ceased completely. Five times the reaction gave slight results, once with cold water, but no results from hot; four times a slight and three times a very marked reaction.

In those cases with marked reaction the operation gave the following result:

I. Very slight edematous swelling, semicircular canal intact, fenestra ovalis empty and open. In a second case the semicircular canal looked dark in its anterior portion. Fenestra ovalis empty, the membranous labyrinth in the semicircular canal and vestibule succulent and dark-colored. In the cochlea were granulations. In a third case (fistula in the promontory), much hemorrhagic fluid in the horizontal semicircular canal; the contents of the vestibule dark red, hearing much lessened.

In cases with moderately strong reaction the result was as follows: First, the horizontal semicircular canal looked dark through the anterior crus, fenestra ovalis empty, in the vestibule a glazed swelling of grayish-red color.

In cases where there is no reaction whatever the horizontal semicircular canal appears dark in the forepart about the ampullar area. Fenestra ovalis penetrable by the probe. The membranous semicircular canal is yellowish-red, in the area

of the ampullae dark brown, and represents here a stout cord. Concerning the contents of the vestibule I could not come to a precise diagnosis.

The above phenomena further show that only the negative result gives a perfect proof; moreover, that we can find with a well-conserved reaction severe inflammatory alterations in the vestibule and semicircular canal, which have often led to fatal meningitis.

The lack of caloric reaction indicates, moreover, a severe affection of the vestibule, but of what kind the changes are, the experiment does not disclose. From the acute hemorrhagic swelling there would usually appear to be an important diminution, but sometimes only a slight one; with chronic glazed swelling, the reaction is often more pronounced. There may be a glazed swelling and even pus in the vestibule, and, notwithstanding, the caloric reaction may still occur in a more or less marked degree; there may be a cicatricial healing process or an ossification of the entire labyrinth, or a complete suppurative decay; there is always the same result—loss of caloric reaction. For the diagnosis of an affection and the functionability of the vestibularis, the experiment is doubtless very valuable, but it discloses nothing of a surgical advantage or of the character of the infection in question.

Do the other labyrinth symptoms reveal anything similar, more important or less so? The remaining symptoms in relation to the vestibularis consist of equilibrium disturbances, spontaneous nystagmus and vertigo, and are very significant, especially in combination with the caloric reaction.

NYSTAGMUS.

As early as in 1893 I described nystagmus as a symptom of labyrinth disease, observed especially when the eyes are directed to the healthy side, and in 1896 I gave a detailed account of a great number of cases showing its frequency, manner of appearance and diagnostic importance. It may often be produced during examination by the application of galvanocautery or catheterization. Since the beginning of 1890 I have made numerous observations on the appearance of nystagmus, vertigo and disturbances of equilibrium after application of galvanocautery. The nystagmus lasts from a few seconds to several minutes. It disappears at the same moment as the vertigo ceases, but a certain weak-

ness or an unsteadiness on the legs remains in some cases for a longer or shorter time.

The reaction is usually very marked. The nystagmus may occur in any form but it is always directed towards the diseased side and is often very intense with extensive, rapid movements. The eyes roll irregularly to and fro, with long oscillations, both rotary and horizontal, so rapid that counting is utterly impossible; the eyes are uncontrollable and can not be fixed on any given object; gradually the eyes get steadier, the movements grow less frequent and smaller, until at last we can count them and at the end they appear only when the eyes are directed to the diseased side; after a few minutes the eye is perfectly quiet again.

This sort of nystagmus is always accompanied by vertigo, which corresponds perfectly to its intensity as well as to the disturbances of equilibrium.

While catheterizing with cold air I have frequently produced nystagmus directed to the side opposite to the healthy one, it having the same effect as syringing with cold water. The sense of cold produces this reaction of the vestibular nerve. This nystagmus is always associated with vertigo. On catheterizing, the irritation is much less than with galvanocautery. Long before Barany's publication, I observed nystagmus following application of the galvanocautery and catheterization with cold air. With the galvanocautery the nystagmus is strong and is directed towards the affected side, while in catheterization with cold air it is directed towards the opposite side.

The appearance of nystagmus and dizziness from pressure upon the auditory canal has long been known; in such a case there is a fistula in the labyrinth wall.

The direction of the nystagmus depends upon the quality or kind of the irritation affecting the vestibular apparatus.

Spontaneous nystagmus is very frequently seen in diseases of vestibular apparatus, generally directed to the healthy side and becoming intense when the eye is turned in the direction of the oscillations. In mild cases it only appears in this direction. It is a sign of greater irritation, if much nystagmus appears in both directions. Slight oscillations of this sort are unimportant.

Nystagmus oscillations always increase if the eyes are

turned in the direction of the oscillations, and they disappear by turning the eyes in the opposite direction.

If nystagmus oscillating towards the healthy ear appears also when looking forwards and to the diseased side, it is a proof of great irritation, of severe infection and of rapid progress of the inflammatory process in the vestibular apparatus.

In addition to the nystagmus oscillations we frequently observe a great restlessness of the eyes. After several slight movements to and fro, which are quite easily distinguished from nystagmus, the eyes rest at a fixed position and from this it is sometimes shaken to the right or to the left.

In severe cases of nystagmus there is an inability to fix the eyes on any given object. The eyes do not remain stationary, but deviate slightly to the other side; in very severe cases they turn to the midline and then return with a sudden jerk to the end position.

There is no doubt that the rapid appearance of nystagmus after application of galvanocautery, after catheterizing with cold air, on pressure of the auditory canal, or after syringing with cold and hot water is symptomatic of labyrinth irritation on that side, being the symptom of an irritation of the vestibularis either from treatment or irritation. With different directions of the movements, the oscillations vary according to the gravity of the irritation.

The spontaneous nystagmus produced by ear suppuration has the same cause; at least in the beginning there is an irritation of the vestibularis, somewhat similar to the use of cold water. In many forms of decay of the vestibularis, however, nystagmus may be looked upon as a symptom of paralysis of the nerve. According to the degree and kind of the irritations the oscillations are directed to the healthy or to the diseased side. At present it is not known why suppuration in the vestibule induces nystagmus in the same direction as cold water does. It is singular that with suppuration in the vestibule, nystagmus is usually directed towards the healthy side.

Experience further shows, that the more acute the process in the vestibule is, the quicker will be the movements of nystagmus. A high degree of nystagmus is produced either by a very severe labyrinth inflammation, or great ir-

ritation in the labyrinth, for instance sequestra, empyema (under compression), or granulations under pressure. There is also a high degree of dizziness and disturbance of equilibrium. Changes in the labyrinth capsule, sequestra, high outside pressure with fistula may also be the cause. On the other hand, there may be a severe disease of the labyrinth without any nystagmus and strong spontaneous nystagmus alone or with only slight degree of equilibrium disturbances is observed in cases where there is no other sign of severe labyrinth disease.

There must naturally be nerves capable of function to produce this nystagmus. It seems doubtful whether nystagmus can arise from decay of the nerve endings or the nerve trunks.

At all events it is important for us to know that generally, but not always, severe nystagmus is a symptom of a severe affection of the labyrinth.

But what is the state if we have but slight nystagmus or none at all?

In many cases where the vestibularis is completely or almost completely destroyed and consequently its function almost eliminated, we still observe spontaneous nystagmus, usually only in a slight degree, sometimes more, and often entirely absent. From my experience, this kind of nystagmus is always directed to the healthy side and apparently produced by the cerebral center or by the other vestibularis.

From the existence of only slight degrees of nystagmus, we can not conclude that there is only a slight affection of the labyrinth, and it would be quite a mistake to conclude that the vestibule is healthy if there is no nystagmus.

Just in those cases where labyrinth infection is latent, where the nystagmus has subsided in consequence of disease of long duration in the vestibular apparatus, the result of caloric reaction is of great diagnostic importance and renders the affection manifest. The disturbed equilibrium usually corresponds with the degree of nystagmus.

Since we are in possession of the convenient and on the whole trustworthy method of caloric reaction, examination on the revolving chair and turning the patient around several times seem superfluous and are no longer used regularly.

The symptoms obtained from the disturbances of the

vestibularis are as already seen numerous and give a sharp clinical picture. The symptoms on the side of the cochlearis are far behind those of the vestibularis in their frequency or in their obtrusiveness. However, there are usually disturbances on the side of the cochlearis, which sometimes are very valuable for the diagnosis.

The symptoms of deafness become characteristic if they develop under our eyes, if we can witness the sudden change of Weber's test, the elevation of the lower tone limit, the lowering of the upper tone limit and the loss of hearing for speech.

The kind and the degree of the equilibrium disturbances give us valuable signs of the intensity of the labyrinth affection, especially for its surgical importance; more so than the lack of caloric reaction, the great value of which consists chiefly in making manifest a latent labyrinth affection. The more severe the labyrinthine symptoms are, the more dangerous is the affection generally, and the more necessary is the opening of the labyrinth.

With the help of the improved diagnostic methods of examination and especially with the assistance of the caloric reaction, we shall generally be able to diagnose the disease of the labyrinth before the operation. This is naturally of great value, for we can begin with the operation well planned in advance.

INDICATION AND PROGNOSIS OF THE LABYRINTH OPERATION.

According to my experience the labyrinth operation is in general not dangerous; the opening of the vestibular apparatus is particularly without danger.

The technic is simple. Danger would only result from penetration of the inner vestibular wall and from injury to the dura in the inner auditory meatus. But this injury is exceedingly easy to avoid when the operation is performed *lege artis*, and when there are present no abnormal conditions such as are seen in the ossification of the labyrinth. Moreover, a highly situated jugular bulb can scarcely signify an appreciable increase of danger to life. Indeed, in consequence of narrow-spaced conditions, a small mastoid process, strong prominence of the lateral sinus, proximity of the cerebellar dura may render the operation difficult to such a high degree that the correct procedure is only with great precaution possible without injury to the adjacent organs.

Moreover, the opening of the cochlea performed *lege artis* appears to be unattended by danger, but having practiced the opening of the cochlea in 23 cases only, while I have opened the vestibule over 100 times, I should not permit myself to make a positive statement upon this question.

It seems certain, however, that in the opening of the cochlea injuries to the adjoining structures, i. e., the dura in the inner auditory canal, occur much more readily, and may be regarded less as technical faults than in the opening of the vestibule.

Further, we have come to the conclusion that infection of the vestibule renders surgical interference necessary much more frequently than infection of the cochlea.

The complications of the deep extradural abscess, of empyema of the saccus endolymphaticus, and of cerebellar abscess, occur only by this localization. The infection invades the vestibular apparatus through the oval window or the horizontal canal. The infectious disease is much more frequent than the cochlear malady, and more extended and more progressive. The cochlear affection when fatal seems to have a slower course. For all these reasons the vestibular apparatus must come first in our consideration of operative treatment.

It will be readily admitted that disease of the entire labyrinth represents a much more serious malady and renders the possibility of extension to the meninges decidedly easier than when only a single portion of the labyrinth is infected. A warning to us are the four cases in which, after a successful vestibular operation, death was produced by meningitis, granulations being scraped out of the vestibule a long time after the operation, or, concurrently with this scraping out, Thiersch's skin transplantation being performed.

These cases demonstrate to us that perfect freedom from danger is not guaranteed even upon the evidence of the safety of the operation; danger to life may still arise from the subsequent treatment. These dangers, however, diminish if they are anticipated, and, accordingly, the Thiersch's skin transplantation, as well as the curetting, must be used with the greatest care, or be entirely avoided.

What indications are to be derived from our observations?

If we appreciate the freedom from danger of the opening of the vestibular portion and consider the dangers resulting from subsequent treatment as avoidable, we should be able to set up the principle, as soon as labyrinth disease has been

diagnosed in one of its principal parts, that the operation is admissible in every case in the face of the dangers which may arise to the patient from labyrinthine disease. I may state here that in this time of observation I have seen ten fatal cases among 190 cases of decided labyrinthine disease in which only the middle ear was operated upon. The labyrinth operation was naturally done in the most severe cases. We have indeed acquired evidence, from a great number of positively diagnosed cases of disease, at least of the vestibule portion, in a great number of cases, the labyrinthine disease does not end fatally, nor in a chronic ailment, but in a spontaneous cure.

In my opinion there is not the slightest doubt that in the majority of these cases disease of the entire vestibular portion has been present, even though single or numerous instances of it may be regarded as circumscribed disease of the semicircular canal. We often found, a long time after the cure, negative caloric reaction. We see from this, without doubt, that the labyrinthine disease, in many cases, ends in spontaneous cure, also that it does not always, in fact, incline to progression, but the cure results slowly and labyrinthine symptoms lasts a long time. The labyrinth operation is permissible in every diffuse infectious disease of the entire labyrinth or of one of its two portions, especially of the vestibule.

The operation should not be performed when it is a question of circumscribed disease in the labyrinth wall or in one of the semicircular canals; when the labyrinthine disease is complicated with infectious processes in the posterior cranial fossa, seldom in the middle one; meningitis serosa, cerebellar abscess; caries necrotica of the pyramid. As a rule this applies to the horizontal semicircular canal.

When can circumscribed semicircular canal disease be diagnosticated and consequently labyrinthine operation be omitted?

1. When the labyrinthine symptoms are not present in excess.
2. When the function of the cochlea is not destroyed.
3. When the caloric reaction is marked, or at least well present.
4. When the fenestra ovalis is intact.
5. When acute middle ear suppuration is wanting, because of the tendency to diffuse labyrinthitis.

Carious processes on the outside of the labyrinthine capsule cannot be a reason for performing the opening of the labyrinth.

Operation is strongly indicated:

I. Where serious disease of the entire labyrinth or one of its two parts is present, usually the vestibule being affected.

II. When in stapes luxation, after 24 hours, the symptoms present are increasing nystagmus, equilibrium disturbances and coated tongue, growing worse. If there is fever, it is then most necessary to operate.

When is the serious disease of the labyrinth or of the vestibule alone?

1. In acute middle ear suppuration every vestibular disease is serious and dangerous, and every fistula in the semicircular canal is to be regarded as a sign of a present or imminent affection of the entire vestibular apparatus.

2. (a) Upon acute appearance of severe labyrinthine symptoms.

(b) Especially in severe labyrinthine symptoms, not arising from pressure of cholesteatomata, granulations, pus on the perforated horizontal semicircular canal, the caloric reaction lacking.

3. When, under observation, the caloric reaction is lacking or diminishing, the function of the cochlea diminishes, the vestibular symptoms become gradually worse.

4. Fever, without other causes.

5. In cerebellar complications in the posterior cranial fossa, especially meningitis serosa.

6. Severe labyrinthine disease is further recognizable during the operation by

(a) Appearance of pus, granulations from the fenestra ovalis or another fistula.

(b) More extensive necrosis, especially at the vestibular wall.

Caloric reaction will be lacking or decreased except in 1 and 2, in which it may be reduced. Its absence, however, is not necessary, since it alone does not prove the presence of a severe affection.

I adhere most decidedly to the indication for operating in severe labyrinthine symptoms, not arising from the middle ear. Only when they have existed for a long period and the vestibular reaction is well maintained, as well as the cochlear function, can one expect the result of the radical operation. I lost a few patients because in spite of severe labyrinthine symptoms I could not make up my mind to operate at once.

METHODS OF OPERATION.

In opening the vestibule I have for fifteen years employed two methods, which I made public in various papers in the years 1893-1900. One method accomplishes the exposure of the vestibule, inside the temporal bone, by following along the horizontal semicircular canal; the other one by removing the posterior wall of the petrous bone from the posterior cranial fossa at the upper crus from the lower, and at the posterior crus from the upper canal, as well as at the posterior crus from the horizontal semicircular canal. Injury to two organs must be avoided—to the facialis in front, which may occur in the first method; to the dura behind, which may occur in the second method.

With a high position of the jugular bulb and the fossa jugularis, a lesion of the bulbus is also possible, and if the fossa jugularis stretches very high up into the temporal bone the bulbus, especially when the opening is made from behind, may be very much in the way of the operation, even making it impossible. A lesion of the inner wall of the vestibule, with injury to the dura of the internal auditory canal, is almost impossible, if the operation is properly performed. But caution is necessary, the wall in part being very thin. Injury to the dura in the meatus would have fatal results. I once saw death result. Opening of the internal auditory meatus is quite possible, if one loses the direction and penetrates above or behind the vestibule into the petrous bone, instead of forwards and downwards into the vestibule.

OPENING ALONG THE HORIZONTAL SEMICIRCULAR CANAL—SEMICIRCULAR CANAL OR TYMPANAL METHOD.

I usually begin the operation by removing the upper half of the horizontal semicircular canal, and thus fix upon the anterior crus of the horizontal canal the limit below which one may not go. By operating carefully it is always possible to preserve the lower wall of the anterior crus; then the facial nerve is protected from lesion. At the posterior crus I remove the whole canal and to keep away from the roof of the vestibule. I also remove the bone under this part of the horizontal canal in the direction sloping down to the back, taking the very greatest precautions with regard to the facialis, which lies in front.

The stapes plate reaches with its posterior margin about the

lateral boundary of the vestibule and takes up a middle position between horizontal and vertical. By this and by the extension of the ampullar extremities of the horizontal and upper canal, the upper portion of the vestibule leads toward the front.

If I penetrate in this way by the anterior crus in the direc- of the canal, and above its lower wall towards the vestibule, and by the posterior crus close to its posterior and lower edge, I have selected a large portion of the lateral wall of the vestibule as the place of attack.

Thus it is easy to open the vestibule either in its posterior lower part, or near the ampulla of the horizontal semicircular canal. According to the pathologic changes, the vestibule is to be opened here or there, in front, above, or at the posterior orifice of the semicircular canal; in cases without fistulae, in the described way, along the horizontal semicircular canal, in the direction of the oval window, parallel with, behind, and a little below the facial bridge. Thus the facialis is surely protected from lesion.

The direction in which we have to seek the anterior wall of the vestibule is indicated not only by the anterior crus of the horizontal semicircular canal, which might lead us astray by its long extended position, but also by the fenestra ovalis, which we can in any case precisely locate by the introduction of a hooked probe. In cases of narrowing of the vestibule by new formation of bone we must expose the front part, at the same time preserving the portion of bone which supports the lower wall of the anterior crus, as a bridge. We penetrate behind and parallel with this bony ridge, inwards, downwards, forwards, in the direction of the fenestra ovalis. If the vestibule is found, the examination with a hooked probe will give a certain opinion as to the size, form and position of the cavity. The lateral wall may easily be felt with the probe, and then neatly removed with a narrow chisel or burr.

If one wishes to obtain information of the contents of the vestibular apparatus, the chisel is to be preferred. To prevent the anterior crus from snapping off, the burr is the better instrument. By using necessary caution and by experience the facialis need not be exposed. It is of the greatest importance to use the chisel as a plane, and to plane off shaving after shaving.

It is of importance to reach the deepest point of the vesti-

bule, to lay bare its floor, to smooth the way to it properly, and to arrange that it may be seen at a glance. By exposing the lower orifice of the inferior semicircular canal we enlarge the cavity of the vestibule downwards, and by exposing the common orifice of the vertical semicircular canal we enlarge it at the back. The burr is eminently adapted to enlarge the vestibule towards the back, either to reach diseased bone lying behind the labyrinth better, or to create below more favorable conditions for wounds, with or without exposure of the dura.

Finally I expose the ampulla from the upper vertical semicircular canal, and then consider the operation completed. Only very rarely do I follow up all the canals, opening them in their full length, starting from the cavity of the wound.

The disease in the canals is usually slight, and after exposure of the orifices into the vestibule, the conditions for healing are specially favorable in them. It is not necessary to expose it further from the fenestra ovalis. Only if the edge of the fenestra ovalis itself is diseased, or the wall of the labyrinth is affected, and an operation on the cochlea becomes necessary, do we consider it expedient to scrape and enlarge the fenestra ovalis with the burr. In general I warn against this, especially against vigorously enlarging the fenestra ovalis towards the back and below, for the bridge which supports the facialis is thus made narrower and necrosis is facilitated. As a rule I employ this semicircular canal method. In fact I consider it the obvious method, in the ordinary forms of labyrinth disease, pointing to the safest way. Any danger to life is out of the question, is impossible.

I should most emphatically reject all attempts to arrive at an opening of the vestibule from the fenestra ovalis only. The widening of the passage is too inconsiderable, and can, in my opinion, not be of sufficient use, even if, by widening downwards to the back, the opening can be enlarged to the floor of the vestibule. The cases which are cured by this method of operation would in all probability have healed quite spontaneously.

ENDOCRANIAL METHOD.

I have much more rarely employed the second method of exposing the vestibular apparatus after removing the posterior wall of the petrous bone and widely opening the posterior, and, when necessary, the middle cranial fossa. I performed this operation for the first time in the year 1895, in the case of a

deep extradural abscess which had penetrated into the labyrinth. In the same year I also indicated the circumstances under which I considered it right to employ this method. In Blau's Encyclopedia I have fully described both methods.

The removal of the posterior wall of the pyramid as far as the labyrinth wall follows the radical operation. Starting from the back, the labyrinth is now removed as far as the middle of the horizontal semicircular canal.

One begins by chiseling away the lower vertical semicircular canal, the posterior crus of the upper one and adds to the removal of the posterior half of the horizontal one. The exposure of the posterior part of the vestibule is easy and without danger. Working at the dura of the cerebellum at this great depth is not quite so free from danger on account of the fixation of the dura here to the aqueductus vestibuli. The dura occasionally tears, fluid runs off, and even if the cases mostly take a favorable turn, one cannot be certain of a good result. I have seen such cases end fatally. In order to expose the anterior portion of the vestibule thoroughly, one must remove some bone behind the anterior crus of the horizontal semicircular canal and parallel to it.

The anatomic conditions at the temporal bone are sometimes very complicated, and abnormal position and development of single parts may render the carrying out of this method simply impossible. Such conditions may be seen where the fossa jugularis has developed extremely high up and immediately behind the vertical semicircular canal high inside the pyramid. The disease of the bone in rare cases goes deeper still, so as to compel one to penetrate at the posterior pyramid wall as far as the porus acusticus internus. It seems to me inadmissible to lay down this as a rule, although it has been proposed. We have to remove that which is necessary, not that which is possible. This endocranial method takes place in all complications in the posterior cranial fossa, and in all diseases of the bone which reach as far as the posterior pyramid plane (floor). Here, also, the horizontal semicircular canal, so easily recognizable, serves best as a means of direction and guidance.

It is desirable also expose the anterior portion of the vestibule in such a way that a sufficient view may be obtained from the auditory canal and the treatment thus carried out. The small portions of bone which impede the view from the side and front may be removed without danger to the facialis.

Besides the entire removal of all diseased matter from the portion of the vestibule, this method has the advantage of being absolutely safe for the facialis. If the exposure of the front portion of the vestibule is not carried out exactly from the back as here described, the enlargement of the entrance of the fenestra ovalis downwards and backwards is indicated.

This method is recommended by Neumann as the standard operation in the opening of the labyrinth, and is generally performed. I cannot commend this plan, for the operation is certainly rendered more difficult by the extensive exposure of the cerebellar dura, and involves the danger of perforation of the dura and imminent meningitis. The more rapid healing is of itself no sufficient reason for the regular employment of this method. Healing would take place even more quickly, if the whole of the upper wall of the pyramid were removed.

The depression of the temporal dura at once causes a quite considerable lessening of the cavity of the operation, but this degeneration does not always mean an acceleration of the healing process. It may very frequently have an extremely detrimental effect.

I endeavor to avoid exposure of the dura, if possible, and consider the preservation of the bony edge of the mastoid an advantage well purchased under certain circumstances, even by a somewhat slower healing process. Still I wish by no means to express the opinion that in this way healing is in general delayed.

The semicircular canal method is, in my judgment, very much simpler and easier. On account of pathologic changes, the construction of the temporal bone may be such that this method is by far the easiest.

In a serious destruction of the horizontal semicircular canal, where it is perhaps only necessary to remove 1 or 2 mm. of bony substance for the complete opening of the vestibule, no one will propose the far more dangerous and troublesome, and also technically difficult operation from behind, and if the facialis should have to be entirely destroyed there is certainly no reason for considering this method the standard one.

But there is yet another objection which I should like to raise at this point, and that is as to the name which has lately been given to this method of operation. Ten years ago it was known as my method, later it was called the Neumann-Jansen method, and now is only known in Vienna as Neumann's method.

Although it is a great honor to me that my method should have been adopted by so distinguished a surgeon, and although believing that the opening of the labyrinth is not a difficult feat for another to have done, I consider it my duty to say that I wish to keep the share that I have had in the invention of this method.

OPERATION UPON THE COCHLEA.

Although formerly I proposed the complete removal of the cochlea, and have often performed it, I have restricted more recently myself to the removal of the promontory and the exposure of the lower cochleal whorl for the preservation of the modiolus. I have grown cautious, having seen one case end fatally in which, after complete removal of the cochlea, the dura in the interior auditory canal was perforated by probing, and discharge of the fluid lasted several days.

In order to obviate this with safety, it is necessary to limit oneself to the removal of the promontory, to the exposure of the lower cochlear whorl round about the modiolus, without injuring the modiolus itself. Should one inadvertently or, owing to destruction in the cochlea, arrive at the bottom of the inner auditory meatus, the probing in this dangerous region, and the opening of the dura, must be avoided. Upon the opening of the subarachnoidal space the cerebrospinal fluid appears, mostly to a considerable extent and of several days' duration.

In order to guard against any possibility of lesion to the subarachnoidal space it seems expedient not to interfere with the modiolus at its base, but to leave it here undisturbed. With these propositions of Richards I entirely concur.

When the cochlea in the interior has become transformed into a bed of sequestra, pus and granulations, the removal can be effected without danger by means of the gouge. I generally perform the operation either with a chisel or a burr, the smallest chisel by preference, beginning with the enlargement of the fenestra ovalis towards the front below. If the burr is used, the mode of operation is very simple; it is introduced into the fenestra ovalis, the bone is chipped off towards the front below at the promontory, corresponding to the lower cochlear whorl.

XI.

PATHOLOGY AND BACTERIOLOGY OF SUPPURATIVE DISEASES OF THE TEMPORAL BONE.

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PATHOLOGY.

The subject of this paper is so very large that only a few of the newer phases of the pathology can be mentioned.

In the temporal bone we have to consider the pathology of cavities lined with mucoperiosteum; cavities of varying size and shape with walls of paper thinness to walls of several millimeters thick; mastoid cells filled with air and cells filled with bone marrow. Underlying the mucoperiosteum, we must consider the pathology of the bone of varying character from soft bone to bone of ivory-like hardness. Within the bone we have muscles and nerves; there is the membranous labyrinth; there are blood vessels and the lymphatics—in fact within and bordering the temporal bone (dura and brain) we have to deal with a great number of different tissues. We also have the pathology of wounds to consider. Why one wound heals readily and another refuses to heal. Again, the pathology of the infant varies from that of the adult, as the anatomy varies.

Let us first take up the pathology within the anatomic middle ear and then pass to the pathology of structures without the middle ear, the latter of which are the so-called complications of suppurative diseases of the temporal bone.

I.—PATHOLOGY WITHIN THE MIDDLE EAR.

We are all familiar with the pathologic changes so well described by Körner, Grunert and Bezold in their text books on acute and chronic suppurative diseases of the middle ear, so we will pass on to mastoiditis. P. T. Hald (*Zeit. f. Ohrenh.*, liii Bd., S. 281) has taken the specific gravity of pus in acute middle ear suppurations. He says if the specific gravity is

over 1045 one should think of an empyema of the mastoid. From 1046 to 1047 and over the diagnosis is assured.

The Pathology of the Mastoid Cell.—The mastoid cell may be pneumatic or diploetic, containing air or bone marrow, so we may have disease of the mucous membrane extending to the bone, causing an osteitis in the one case or a disease of the bone marrow or osteomyelitis in the other case. Pneumatic cells may be divided by mucous septa, as seen in Fig. 1. In these air cells there are usually several processes going on singly or all at the same time: First, there may be a lack of air; a partial or complete vacuum with its resulting passive hyperemia and transudation. Second, there may be bacterial invasion of the mucoperiosteum with active hyperemia and round cell infiltration (Fig. 2); in infants there may be swelling of myxomatous tissue, so well illustrated by Preysing in "Otitis Media der Säugling." By this process the air space may be nearly occluded. The pavement epithelium is loosened from the submucosa and the exudate may be so great that all of the air space is obliterated, and then, third, pressure begins its destructive work. Pressure shuts off the blood supply and kills the mucoperiosteum and secondarily the bone. The pressure is the greater since it is confined in a cavity nearly surrounded by bone. Besides the pressure we have the toxic effects of the bacteria, which depend on the virulence of the bacteria.

Fig. 3 shows a mastoid cell in an acute suppurative disease of the mastoid, operated on by Dr. E. A. Crockett, of Boston. The mucoperichondrium is strongly infiltrated on the inferior side with necrosis of the mucoperichondrium and hemorrhage into the pneumatic space. Preysing shows in his book some mastoid cells with the vessels in the submucosa filled with thrombi.

Figs. 4 and 5 show pus in cells of a rachitic child who, according to clinical diagnosis, died of diphtheria. The mastoid cells are filled with pus. The mucoperiosteum has been destroyed. Fig. 6, a portion of Fig. 5, shows the destruction of the bone by osteoclasts. The milder types of suppuration with formation of granulations are shown by Preysing. Granulomata may be protective or obstructive in their action, depending upon their position.

Dr. Coutade writes of three cases of *serous mastoiditis*

where 15 to 20 cc. of a yellow lemon-colored serum containing some mucus was found at operation. These patients had meningeal symptoms.

Tubercular mastoditis in children. Henrici says less than one-sixth of all mastoiditis in infants are tubercular. There may be a hematogenous primary osseous infection with normal state of the tympanic cavity and antrum, with formation of sclerotic bone and granulations in mastoid cells the size of small peas, and fistulae. The swelling of lymphatic glands over the mastoid is not characteristic of tuberculosis.

Isemer (*Archiv. f. Ohrenh.*, lxvii Bd., S. 97, 1906) reported in detail forty cases, and stated that tubercular mastoiditis occurs in the first half of the years of childhood more frequently than usually supposed; about thirteen per cent of all cases of mastoiditis were tubercular, agreeing with Henrici.

About half of these tubercular mastoiditis cases are primary, that is, originate by the way of the blood channels; the other half are secondary, mostly progressing from tubercular diseased lymphatic tissue in the nasopharynx. The surest proof for a tubercular disease of the mastoid is obtained from microscopic investigations of the diseased parts; less certain are animal experiments. Haike described three cases of primary tuberculosis and two of secondary from the lungs of infants. Paralysis of the facial nerve occurred after a few days with rapid destruction of the temporal bone. In three weeks there were destruction of the ossicles and suppuration of the labyrinth.

In *diabetes* Grossmann says (*Beitr. z. Ohrenh. Festsch. zu Lucae's* 70 Geburtstag, S. 353): "The mastoid is more readily diseased than in those patients without diabetes. In otitis media with diabetes 72.7 per cent had mastoiditis, while of cases without diabetes 56.3 per cent had mastoiditis. The cause is due to the lessened resorptive activity and greater frailty of the mucous membrane; a result of early arteriosclerosis, which occurs in most diabetics together with the altered composition of the body fluids. There is no special form of diabetic mastoiditis."

Wolff (*Zeitschr. f. Ohrenh.*, liv Bd., 1907, S. 322) wrote an interesting paper on *recurrent mastoiditis*. For example, following acute middle ear inflammation, a mastoiditis occurs with formation of abscess; the disease heals after operation com-

pletely, but after a longer or shorter intermission in consequence of a new otitis an abscess quickly forms. This process may be repeated several times. The intermission varies from four months to three and one-half years. I have had two cases where there was a recurrence after mastoid operation due to trauma, one playing football and the other struck in playing. Both healed quickly with great retraction of the scar.

Bezold's mastoiditis and *gravitating abscesses* from the mastoid. I have seen a few cases of acute mastoiditis where on operation, the antral cells were macroscopically normal, with the tip cells filled with pus and a perforation extending into the neck muscles. The pus may pass from the tympanic sinus internal to the facial canal to the tip, or the pus may take Voss's route in operative work on the jugular bulb, the infection not spreading by way of the antrum.

Engelhardt (*Zeitschr. f. Ohrenh.,* liv Bd.)in an article on otogenous gravitating abscesses gives eight ways in which retropharyngeal abscesses occur, as follows:

1. Through direct rupture of an osteomyelitic or tubercular focus from the diseased petrous bone to the throat (Muck, Klug and others).
2. Through spreading of inflammation from the middle ear along the tensor tympani or progressing directly in the tissue around the tube (Kien, Haug, Blau, and others).
3. Through the floor of the antrum to the anterior wall of the auditory canal spreading to the retropharyngeal space avoiding or not avoiding the articulation of the jaw (Kien).
4. From the tympanic cavity with resulting perforation into the articulation of the jaw, and from there spreading to the retropharyngeal space (Grüber).
5. From subdural (DeRossi) or extradural (Bramstein) suppuration from the posterior fossa of the skull with resulting perforation.
6. Spreading of sinus thrombosis through the superior and inferior petrosal sinuses to the basillar plexus; secondary suppuration of the same with formation of a retropharyngeal abscess (Schultze).
7. From peribulbar abscesses which spread horizontally under the base of the skull resulting in perforation of the retropharyngeal space (Jansen, Grunert).
8. Through the lymphatics with secondary suppuration of

retropharyngeal glands after acute suppuration of the middle ear (Most).

II.—PATHOLOGY OF STRUCTURES WITHOUT THE MIDDLE EAR.

I can quote only a few facts relating to labyrinthitis. There are many disputed points in this field of otology.

Macroscopic reports of labyrinth suppuration are of little worth; they must be microscopic.

Labyrinthitis in epidemic cerebrospinal meningitis. Göerke (*Verhandl. d. Deutsch. Otol. Gesell.*, xv, S. 227, 1906) found on examining nineteen temporal bones that the internal ear was affected seventeen times; changes were only found in the basal turn of the cochlea. Eleven times infection spread along the acoustic nerve, once along the aquaeductus vestibuli, and three times through the aquaeductus cochleae.

Oscar Wagener (*Archiv. f. Ohren.*, lxviii Bd., S. 272, 1906) considers the aquaeductus vestibuli as a path for infection to pass from the vestibule to the brain. E. P. Frederick in his book, "The Suppuration of the Labyrinth of the Ear," 1905, considers the formation of an empyema of the sac as possible only in extensive suppuration of the labyrinth and only attributes to it the importance of an extradural abscess. Wagener found in the literature twenty-eight cases which had been autopsied; only three had been studied microscopically. In all these cases the labyrinth was found diseased. Politzer reported the only case where there was no doubt. Wagener concludes that the migration of pus from the labyrinth into the sac by way of the aqueduct of the vestibule is excessively rare.

Göerke says that suppuration of the labyrinth extending to the dura can only pass through the nerve canals and the aquaeductus cochleae. The ductus endolymphaticus quickly fills up with newly formed connective tissue. Lange (*Beiträge zur Anat. Physio. Path. u. s. w. des Ohren. der Nase und des Kehlkopf.*, Band 1, H. 1-2, Jan. 20, 1908) found in three cases of severe labyrinth suppuration necrosis of the aquaeductus vestibuli to the aperatura externa, then normal cells and normal saccus endolymphaticus.

Next, we may consider extradural abscess or so-called epidural abscess and subdural abscess or intrameningeal abscess. *Extradural abscess* is a collection of pus between the temporal

bone and dura mater. According to Citelli (*Archivio italiano di otologia, etc.*, Bd. xviii, H. 2, S. 151) on otitic extradural abscess he came to the following conclusions:

1. Extradural abscesses are caused as often by acute as by chronic suppurations of the ear.

2. They occur as often in the posterior as in the middle fossa. Grunert says most often in the posterior fossa.

3. True extradural abscess is the rarest of such suppurations.

Subdural Abscess.—A collection of pus between the inner surface of the dura and the arachnoid. Pure subdural suppuration is rare since before pus or pathogenic germs have time to form an inflammatory agglutination on the inner surface of the dura they generally spread over the brain and develop a diffuse suppurative leptomeningitis.

Perisinuous abscess generally forms before sinus thrombosis but may form after sinus thrombosis, causing a renewed temperature and phlebitis.

Kramm (*Zeit. f. Ohrenh.*, Bd. liii, H. 1-2) describes obliteration of the sinus by compression and phlebitis by extradural abscess without an inflammatory thrombosis in children. Leutert does not believe this.

v. Uchermann (*Archiv. f. Ohren.*, Bd. lxxi) on otogenous pyemia and otogenous infectious sinus thrombosis makes the following distinction:

"In otogenous pyemia, so-called, there is no formation of thrombi and fever is caused through absorption of pyogenic bacteria and their toxins.

The metastases are always in the covering of the connective tissue and mucous cavities or in the joint cavities, sometimes in the pleural cavities, not in the lungs. In infectious sinus thrombosis one finds inflammation in the sinus wall with partial (parietal) or complete (obstructing) formation of thrombus. The fever is not necessarily pyemic and becomes so only through suppuration of the thrombus mass and through metastases of the parts to the kidneys, spleen, and to the lungs. Otogenous pyemia may start from acute suppurative processes in the tympanic cavity and mastoid cells, sometimes from the labyrinth. Infective sinus thrombosis is mostly from chronic processes. These forms are purely typical. In both forms the picture may change according as the fever has more the char-

acter of a poisoning (toxemia) than a pyemia (bacteremia). Such cases are mostly septicemic eventually septicopyemic. In the mixed atypical cases, the sinus thrombosis is parietal. Hessler collected 357 cases of sinus thrombosis, 192 on the right side and 165 on the left. Körner gives as a reason for the greater number on the right side the fact that, as a rule, the right sigmoid fossa is deeper in the bone than the left, and therefore there is a greater surface to be attacked in case of infection. Especially is this the fact when the mastoid is fully developed. Then again Dr. G. D. Cohen-Tervaert (*Archiv. f. Ohren.*, Bd. lxxiv) on sinus thrombosis and jugularis hypoplasia says that Linser found in 1,022 skulls twenty-nine cases of hyploplasia or 3 per cent of all cases. Hypoplasia is five times more frequent on the left than on the right. There may be hypoplasia of the jugular vein and not of the sinus.

Uchermann in Christiana Hospital in thirteen years found thirty cases of otogenous pyemia and infected thrombosis, 9 in children, 21 in adults. The primary disease of the temporal bone was acute in eight cases and chronic in twenty-two cases.

Hessler found in 130 cases, 31 acute and 99 chronic; about the same relation.

In 18 out of the 25 cases reported by Uchermann where sinus phlebitis with formation of thrombus could be proven the suppurative mastoiditis extended to the sinus wall where perisinuous abscess was found in six cases; in seven cases no apparent macroscopic direct communication could be established.

In all five cases of otogenous pyemia without apparent sinus thrombosis, there was no direct union between the suppurative mastoiditis and the respective sinus wall. In one case the pyemia was caused by complicating suppurative labyrinthitis.

In 24 of the 25 cases with thrombus formation the thrombus was situated in the sigmoid sinus, some extending from there posteriorly to the transverse sinus, and some inwardly in the inferior petrosal sinus, and again inferiorly to the bulb of the jugular vein and in the jugular vein itself. In the 25th case, the sinus thrombosis had its seat in the venus plexus around the foramen magnum and further to the occipital sinus, to the torcular, where there was an abscess, and over to the marginal sinus to the jugular bulb. The bulb itself, as also the sigmoid

sinus, is free and the point of entrance according to ear diseases (pus in the tip of the mastoid process) is a subperiosteal, suboccipital abscess. In one of the 24 cases the thrombotic formation and softening extended to the torcular herophili to the opposite transverse sinus. The posterior half was filled with pus and the remainder filled with normal blood clot to the right superior bulb of the jugular vein where again there was a pus focus. Such extensions of thrombosis to places where the blood streams are slower or form a rotary motion is also mentioned by Körner. Sinus thrombosis in the bulb of the jugular may be developed by bacteria which pass through the sinus wall higher up and wander into the blood current and not as usual pass in from direct disease of the bone on the floor of the tympanic cavity (Leutert, Körner-Muck)."

"In most cases the cavernous sinus is attacked through extension from the petrosal sinuses or of the venous plexus around the carotid. Through perisinuous abscess and phlebitis of the condyloid emissary veins, abscess formation may occur on one or the other side of the condyloid process around the foramen magnum.

In one instance Jansen (*Archiv. f. Ohren.*, Bd. xxv) reported a case of phlebitis of the mastoid emissary vein extending from a parietal thrombus of the sigmoid sinus.

In the cases where no direct union between the focus in the mastoid process and the diseased sinus wall could be seen, often separated by ivory-like bone, Körner assumes that here also an osteophlebitis is the medium from the small veins of the diploe to the sinus wall.

While the sinus wall in pure otogenous pyemia is normal, thin and of a bluish color, in sinus thrombosis it is more or less thickened, often uneven, greyish or yellowish and of a dirty color. Often one finds it sunken in or compressed by pus and granulations. The thrombus in the beginning is parietal and so impossible to feel. If thrombosis is complete then the sinus feels hard like a cord. The walls may grow together, or the thrombus organize, or the thrombus may rapidly break down and suppurate. The sinus wall may ulcerate in one or many places. If an incomplete thrombus lies under the ulcerated spot, bleeding or aspiration of air may take place. The disintegration of the thrombus does not always take place at the point of infection, but may be further back in the transverse

sinus or down in the bulb. Sometimes a fistula forms acting as a drainage tube from the lumen of the sinus.

In six of the twenty-five cases brain abscess accompanied sinus thrombosis."

Postoperative sinus thrombosis is not spoken of in the literature. I have seen one case in which the superior petrosal sinus was wounded resulting in sinus thrombosis and death.

Felix Modowski (*Archiv. f. Ohren.*, Bd. lxxiv, S. 335) writes on the pathology of sinus thrombosis, showing microscopic sections of different parts of a thrombus. The thrombus was organized completely in the left transverse sinus filling it with connective tissue. In the longitudinal sinus the central portion of an organized part had softened.

In general sepsis from the ear, W. Kümmel speaks of neuritis or perineuritis of the nerve roots passing through the jugular foramen especially the glosso-pharyngeus causing pain on swallowing.

W. Cozzolini regards paralysis of the abducens of otitic origin as more of a toxic origin than a circumscribed leptomeningitic focus. Gradenigo believes the paralysis is caused by circumscribed meningitis at the apex of the petrous bone. Transmission of infection from the middle ear is not through the labyrinth, but perhaps by way of the spongy tissue around the capsule. Geronzi believes it to be a reflex paralysis of the neucleus of the abducens.

Kashi (*Archiv. f. Ohren.*, Bd. lxx, H. 1-2) reports 15 cases in infants of otitic dyspepsia with enlargement of the liver. The spleen was enlarged only three times. Pus gets into the digestive tract from the middle ear through the tube. Hartmann and Preysing have described digestive disturbances in infants, as also has B. Gomperz in his book on "Pathology of Middle Ear Inflammation in Infants," 1906. After operation on mastoiditis complicated with syphilis patients are liable to have otogenous pyemia with no visible involvement of the sinuses.

Brain Abscess.—Okada states that in 10 per cent of cases of cerebellar abscess, death occurred before any symptom of intracranial disease was indicated. In 42 per cent the diagnosis was impossible on account of other intracranial complications.

In most cases of brain abscess the bone disease in the middle ear extends to the dura. In about one-half of the cases the

dura appears diseased. The diseased dura is nearly always adherent to the overlying brain surface and between the adherent part and the brain abscess the brain substance is a few millimeters thick. Brain abscesses may be single or multiple. Voss (*Zeit. f. Ohren.*, xli, S. 223) believes that the beginning stage of an otogenous brain abscess is a hemorrhagic encephalitis, which goes on to suppuration.

In a compilation of 645 cases of otogenous brain abscess by Heimann,

456 were in the cerebrum, or 70 per cent.
188 were in the cerebellum, or 30 per cent.
1 was in the pons.

Brain abscess occurs three times as often in men as in women, mostly between the second and third decennium.

457 were caused by chronic middle ear suppuration, equal to 80 per cent.

113 were caused by acute middle ear suppuration, equal to 20 per cent.

Otitic Leptomeningitis.—Leptomeningitis is caused by brain abscess, sinus thrombosis, labyrinthitis, unskilled operations as extracting foreign bodies from the middle ear, curetting protective granulations from the tympanic cavity, etc. Zeroni collected 40 cases of post-operative meningitis from the literature and that is far short of the real number. Acute suppuration is quite frequently complicated with meningitis, as is chronic suppuration. From the labyrinth the pus flows through the internal auditory canal and the aqueductus cochleae. Diffuse meningitis gives a positive result for lumbar puncture, i. e., pus, and pathogenic organisms.

Meningitis Serosa.—The cause is not known whether a collateral edema or a beginning circumscribed suppurative leptomeningitis, or whether it is of toxic nature. It was first described by Quincke.

Zeroni (*Archiv. f. Ohren.*, Bd. lxxiii) speaks of *wounds after mastoid operation* which refuse to granulate but keep up a discharge. Sometimes when the diseased focus is curetted the wound granulates and fills up; again, after curetting, the wound only becomes larger and the epidermis from the edges of the wound grows in, since there is no support by granulation under the edges of the wound.

After operation on the mastoid process, regeneration of the

bone almost never occurs. Zeroni has examined histologically two cases in which radical operation had been done, one six weeks and the other a year after operation. In the first case there was no apparent new formation of bone. In the latter case there was a thin layer of new bone found, but no osteoblasts were found, so the process could not be studied. Spongy bone has the least inclination to regenerate. In normal cases the wound cavity fills with granulation tissue quickly and forms a hard, fibrous cushion which takes the place of the bone and acts as a protection to any deeper parts as dura. This granulation mass may grow from the bone and also from the edges of the skin wound. Granulations spring from the endosteum of the Haversian canals.

Zeroni describes two types as follows:

First, those in which the granulations develop in great abundance from the edges of the cut wound and so cover the opening, at the same time the process occurs in subcortical layers. The new-formed tissue lies in the cavity and joins the adjacent part as soon as the tampon is removed, forming a beautiful firm, small scar, solid and resistant throughout.

Secondly, in other cases the granulations seem to grow more from within. The filling of the cavity is concentric. The skin edges remain in the position in which they were left after operation. As soon as the underlying cushion has attained sufficient thickness, the skin takes part in the healing process, sending out a thin layer of epidermis. The result in such cases is nearly always a more or less deep depression over the mastoid.

Since the granulations seldom reach the level of the outer skin and because the subcuticle layers fail as in the first type, the skin over such a depression remains thin, and is at first very sensitive. The epidermis may die and a superficial ulcer may form. Later the supporting tissue becomes firm, and improvement takes place, but in consequence of retraction of the connective tissue, the depression grows deeper than ever.

There are many variations from these two types. Sometimes both types take place in the healing process.

The first type is seen most frequently in small wound cavities, the second in large wound cavities, made in laying open extradural and perisinuous collections of pus when an extensive resection of the cortex is necessary to give free drainage.

The edges of the wound must remain open for a long time. Even these large cavities may gradually fill up and form a small smooth scar. In these cases, after a commencing good proliferation, suddenly there is a cessation of the process of healing and the external wound comes to a standstill, remaining wide open and the epithelium grows in to meet the deep granulations.

In the radical operation we used to pack to keep granulation tissue down and it was always a fight. Now we are told to leave all packing out after the first dressing. Sometimes the young granulations are killed after a simple mastoid operation by a too firm packing. The blood supply is cut off and necrosis may result.

BACTERIOLOGY.

Otitis media was first divided into two groups by Zaufal, the so-called genuine otitis and secondary otitis. The genuine otitis occurring after nasopharyngeal inflammation, or tonsillitis or inflammation of the upper respiratory tract. Secondary otitis media occurs after the acute exanthemata and is generally caused by streptococci, but in a number of cases examined by Kümmel the secretions were sterile. Kümmel calls these sterile toxic inflammations.

Kümmel-Süpfles' (Transactions of the German Otological Society, 1907) results on the bacterial investigation of acute middle ear inflammation in Heidelberg were as follows:

"1. Sixty-six per cent were caused by streptococcus pyogenes, about 17 per cent by the streptococcus lanceolatus, about 11 per cent by streptococcus mucosus, and finally about 6 per cent by the micrococcus pyogenes aureus and albus. These results correspond with Denker's investigations.

2. The reason why the streptococcus lanceolatus in the literature on the subject was greatly increased over the present results was that the streptococcus pyogenes and mucosus were not differentiated by the early investigators.

3. The greater number of cases caused by streptococcus lanceolatus occur in the first ten years of life (76 per cent). This is so to a less extent of streptococcus pyogenes; first ten years 44 per cent, the second ten years 22 per cent.

4. The course of the otitis depends on the nature of the inflammatory excitor, in so far as an operation is never neces-

sary in otitis with micrococcus pyogenes aureus and albus, otitis with streptococcus lanceolatus only rarely and under certain conditions in childhood. In otitis with streptococcus pyogenes, operation was necessary in the proportion of 1-3. Twenty-three cases out of 96, otitis with streptococcus mucosus about 1-1.

5. The staphylococcus otitis inclines, so far as the small material allows conclusions, to run a protracted course, and it is possible that the frequent mixture of staphylococci with other inflammatory excitors may be the cause of their protracted course. It is one cause out of others which tends to chronicity,

6. The chronicity of an acute otitis is not necessarily the result of the presence of staphylococci.

7. That the otitis with streptococcus lanceolatus, described "cyclic course," could only be observed in such cases which showed beside the otitis still additional localizations, probably by the same inflammatory excitor.

8. That streptococcus mucosus appears, as with the experience of others, to have a great capability of destroying bone and often appears late in the disease, sometimes even when the otitis has gotten well. This peculiarity was wrongly stated to be caused by the streptococcus lanceolatus by earlier investigators, because they had no means to differentiate it from the streptococcus mucosus.

9. The course that the disease takes depends not alone on the kind of inflammatory excitor, but also on the peculiarities which lie in the patient himself, of which we only know the smallest part.

10. One of these peculiarities is the individual nature of the union between the epitympanum and mesotympanum. A free union between the two gives a better prospect of the otitis getting well without operative procedure. The difference in the course of the disease between the so-called epitympanal and mesotympanal otitis is brought out most clearly in cases caused by streptococcus pyogenes, in so far that almost all of the cases which come to operation are of the epitympanal type. A sure pathologo-anatomic support for this separation fails as yet.

11. Moreover, it appears as if the way of invasion of the inflammatory excitor according whether it is termed a salpingogenous or hematogenous otitis, influences the course of the disease, in so far as the hematogenous infection presents more

the picture of a severe general disease and therefore the cavities of the mastoid process take on a less intense sympathetic affection. Our knowledge in relation to the way of invasion of the inflammatory excitor is still, however, insufficient.

12. So-called secondary otitis can apparently exist sometimes without participation of microorganisms, apparently through toxic action and the otitis either quickly heals without the entrance of organic inflammatory excitors or subsequently become infected with such and then runs as other cases of otitis."

Denker (*Verhandlung der Deutch. Otol. Gesell.*, 16 Versammelung) on bacterial investigations in acute middle ear suppuration examined 29 cases of acute empyema of the mastoid process. The diplococcus pneumoniae wholly failed throughout the 29 cases, although 9 of the children had pneumonia. Kümmel-Süpfle found pneumococci only in two cases where the pus was taken from the mastoid.

Wittmaack (*Deutch. med. Woch.*, Nr. 31, 1906) found that staphylococci were only found in the secretion when the secretion had continued several days, therefore staphylococci infection is probably secondary.

Chronic otitis is due to those bacteria which cause a protracted course as streptococcus mucosus and in cases of mixed infection.

Proteus vulgaris is rarely mentioned in acute suppuration. Lauffs (*Archiv. f. Ohrenh.*, Bd. lxx, S. 187) found proteous vulgaris often accompanying cholesteatomata. It often destroys the cholesteatomata and then this putrid bacillus has a good chance to infect the cranial cavity. This bacterium generates sulphuretted hydrogen, ammonia, and indol.

Brieger (*Verhandlung der 7 internat. otol. Kongress*, 1904) said that microorganisms within the middle ear space which are saprophytes could develop under favorable circumstances where the air was occluded as in the labyrinth or arachnoid space and become virulent, causing meningitis.

Rist (*Zentralblatt f. Bact.*, Bd. xxx) says as a result of numerous investigations that he has never found a single fetid suppuration without finding anaerobic bacteria. Franz Kobrak (*Archiv. f. Ohrenh.*, Bd. lxxiv) has an interesting paper on the importance of anaerobic bacteria for the existence of sinus thrombosis and reports two cases.

Pseudo-diphtheria bacilli are more frequently found in chronic than in acute inflammation. They can only be differentiated from the true diphtheria bacilli by experiments on animals.

Lermoyez believes that there are osteophile streptococci.

H. I. Wolff (*Zeit. f. Ohrenh.*, Bd. liv) on the cause of recurrent mastoiditis gives three methods by which the mastoid may become infected:

First, the retention of latent germs in the mastoid process.

Second, a new infection of the mastoid process from the tympanic cavity by virulent germs.

Third, a new infection by mild virulent germs which can cause abscess due to the lessened resistance of the tissue after the first disease and operation, if at the same time there exists a bad general condition or a disposition to otitis.

Ernst Leutert (*Archiv. f. Ohrenh.*, Bd. xiv) in a paper on "The Diagnosis of Otitic Sinus Thrombosis by Means of Bacteriologic Investigation of the Blood," comes to the following conclusions:

"1. The presence of streptococci or other microorganisms occurring in the blood of the sinus in considerable number and at the same time sterile blood in the arm indicates a diagnosis of sinus thrombosis.

2. The diagnosis is sure if the blood in the arm gives a positive result. In cases of positive results in the blood of the arm the prognosis is unfavorable since it must indicate a lessening of the bactericidal strength of the blood.

3. If the sinus blood is investigated alone and a moderate number of pyogenic microorganisms appear, then there is thrombosis unless taken shortly before the patient's death, in which case other septic disease may flood the whole blood system with microorganisms.

4. The negative result of the bacteriologic investigation of the blood in the vein of the arm proves nothing against the presence of a septic thrombosis, for in recent cases the microorganisms are killed by the bactericidal action of the blood.

5. The condition of microorganisms getting into the blood through permeability of the sinus wall. If sinus phlebitis is the first stage of thrombosis these cases must be studied further, since in such early cases the unweakened bactericidal action of the sinus blood has given negative results or only rarely colonies. The number of cases is too small to draw conclusions."

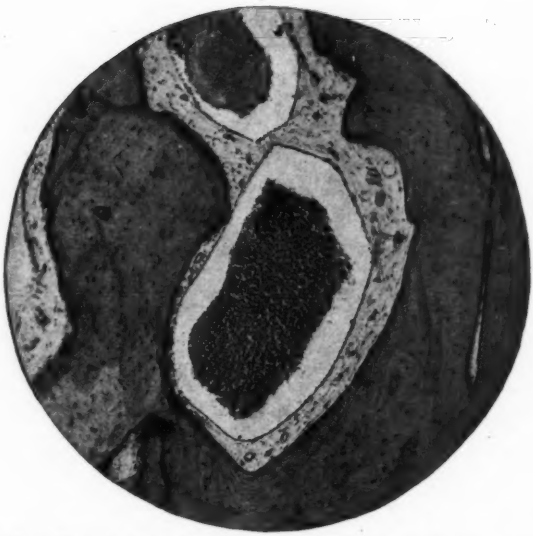

FIG. 1.—Pneumatic cell of mastoid divided by mucous septum.

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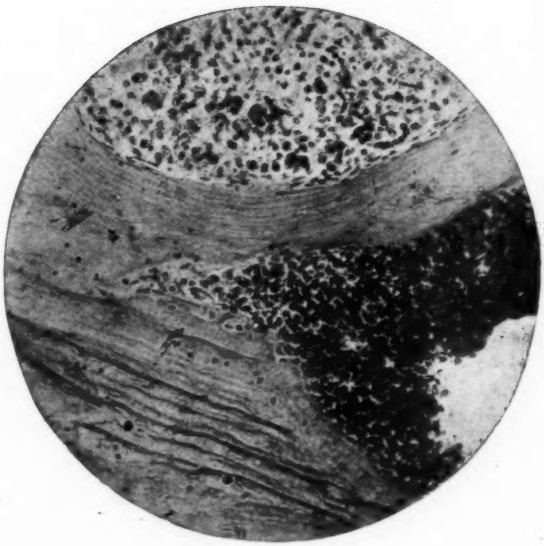

FIG. 1A.

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FIG. 2.—Round cell infiltration.

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FIG. 3.—Infiltration; hemorrhage; necrosis.

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FIG. 4.—Pus; death of mucoperiosteum; caries.

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FIG. 5.—Mucoperiosteum destroyed, showing pus in mastoid cells and necrosis of bone.

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FIG. 6.—Destruction of bone by osteoclasts.

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XIII.

OPERATION FOR BRAIN ABSCESS OF OTITIC ORIGIN.

By J. A. Stucky, M. D.,*

Lexington, Ky.

I wish to apologize to the section for not having prepared a formal paper with some care, but really there is a good excuse back of it. Those of you who have seen the last issue of *The Laryngoscope* will find in that journal, if you care to read it, some of my ideas on this subject. There are some points I want to go into with a little more detail. My topic is "The Operation for Brain Abscess of Otitic Origin."

In submitting a paper on this subject, the supposition is that the diagnosis has been made and that a brain abscess is present; and yet the diagnosis is so often uncertain, on account of the absence of focalizing symptoms and pathognomonic indications that it is well to pause here and consider them carefully, I very much doubt whether in many cases we have any absolute indications of brain abscess. In every case of chronic suppuration of long standing, in which there is marked evidence of septic poisoning, we should be prepared in doing a radical mastoid operation to deal with an extra- or subdural abscess. This point I want to emphasize. In chronic abscess sometimes the abscess is encysted and few symptoms exist. The most prominent of those of an abscess of the brain may be pain in the head, vomiting, with little or no nausea.

I have had only twelve or thirteen cases of brain abscess and I wish to refer to the two most frequent symptoms, and mention the treatment I have followed in these cases. There was a slow pulse and a normal or subnormal temperature. One case upon which I operated on three days ago had a temperature of 96°, and a pulse of 63. The child was twelve years of age. She had an occasional rigor, with some intraocular changes, choked disc, but these symptoms are not reliable. The discharge from the ear had stopped suddenly two weeks ago and the child began to decline. Cerebration became slow, and she had some of the symptoms that were

*Stenographic report of paper.

described by Dr. Layman this afternoon in connection with his case. Mental apathy was marked. I found occasional paralysis of certain groups of muscles. Fortunately, we have in lumbar puncture in these cases a valuable aid in diagnosis. If the fluid withdrawn shows marked infection, we are justified in supposing that we have a brain abscess, if in addition there are any marked meningeal symptoms.

One peculiar feature about the cases I have had is the description of the pain given by the patient. Several have said it is so deep that they cannot find it. There is no pain apparently over the mastoid, but they describe it as being "away down in the ear, so that you cannot touch it; it must be an inch or two in there," and it may be an occipital or temporal pain. I have found in these cases, notwithstanding the experience of Dr. Day, that the differential blood count was most valuable, and I have had examinations made frequently, every eight or ten hours, especially in the fulminating cases.

The prognosis, of course, is to be guarded. Usually it is unfavorable.

Of all cases we have to deal with, those in which we suspect brain abscess are the ones in which we ought to "carefully box the compass," so to speak, and take our bearings. We should consider most carefully the excretory and secretory condition of the patient and the powers of resistance—a point which I emphasized in my address at Louisville. Indican in the urine should be the cause for more anxiety than the presence of either albumin, sugar or casts, and in all these chronic suppurative conditions of the ear, I know of none in which intestinal metabolism is more interfered with or where the vicious circle gives us more trouble. I know of nothing where peristalsis is so completely paralyzed as it is in these cases. You may treat it with purgatives, but what will purge a patient ordinarily will not purge a patient with meningeal irritation or brain abscess; you can give them ten times as large a dose. So I am very careful to make a special effort to get elimination and secretion started before beginning any operation where I expect to go into the brain.

The method of operating is a little different from that usually described. First, I do as thorough a radical tympanomastoid exenteration as is possible for me to do; then I remove

all the tegmen antri and tegmen tympani until I get the floor of the middle fossa well exposed; as complete an exposure as desired may be secured without making a counter opening with the trephine in the squamous portion by widening it until it is sufficiently enlarged. If the dura is discolored and bulging, I open with Bier's knife at the point where there is the most distension, and then, instead of searching with a knife, I invariably use Jackson's searcher. I believe this is safer than using the knife. The different parts of the brain may be penetrated to any depth and opened, and if there is any pus there, it will escape. The incision may be enlarged, if necessary.

In this connection I wish to speak of a very important point which Sir Victor Horsley presented to the British Medical Association a year ago, about which I have grown enthusiastic. His method, which I use in all radical operations in which I expose the sinus or dura, is the almost constant use of hot normal saline solution, at a temperature of from 115° to 120°, never lower than 115°, never higher than 120°. Here is where operative technic counts for more than in any other operation in surgery. Every man and woman connected with it has something to do, and should know how to do it, and when to do it. One nurse takes care of the normal saline solution and must have nothing else to do. The thermometer is always in the douche bag. If the water is below 115°, it does not control hemorrhage; if above 120°, as Sir Victor Horsley has demonstrated, there will be some little reaction, but between 115° and 120° all oozing can be effectually controlled. In addition to this, all soft parts are kept moist and clean. All bone dust and all exudative material are washed out.

After making an incision and beginning exploration, I practically operate under a small stream of water. The hot saline solution is allowed to run from a medicine dropper, attached to the douche bag, over the wound, and the parts are always warm, moist, and clean. If an abscess is found and the incision is enlarged, I do not believe in making any exploration with the finger; if any cheesy particles of brain matter are found they are to be clipped off carefully or sponged away. The sponges should always be wet with the normal saline solution.

After finding the abscess and evacuating it, we should be extremely careful in washing it out. The force of the normal

saline solution should not be any more than enough to throw a stream six or eight inches. I do not hesitate to put a pipette or medicine dropper into the opening and irrigate it until it becomes clean.

I think the most important point to be observed after the operation is the after-dressing, which should maintain constant and thorough drainage. I have ceased to use the cigarette drain or any gauze. I always have on the table three sizes of tubes of flexible rubber, perforated size (see figure). These I cut to the required length. If it is a sphenotemporal abscess, the tube should be placed in the opening through the dura, with simply a little drain of narrow wick of gauze in them, but not enough to plug it up. If in the posterior fossa it should be placed in the proper position, and let it come out back of the ear. The other tube goes up through the auditory canal just as I drain in the radical mastoid operation. A dressing of bichloride gauze is then applied and moistened every four hours with the 1 to 3000 or 5000 solution of mercury bichlorid. The dressing is changed every twenty-four hours under the same aseptic precautions as those under which the operation is performed, because the moment the gauze in the tube becomes saturated or a little clot of blood forms in it, it ceases to act as a drain and becomes a cork. The gauze is put inside the rubber tube and allowed to project, on the order of a siphon. One of the chief things is the thorough drainage we get, and this is maintained by daily dressing the wound. In my first few cases I left the dressing on for two or three or five days, and I believe some of the bad results I had were due to that. I treat my radical operation cases on the same principle. Next to the sponging and drainage in the operation I believe that the quantity of the anesthetic cuts a great figure. It is astonishing how little of the anesthetic we can get along with in these brain cases and in radical mastoid operations. I have had patients held for an hour and a half to two hours on surprisingly small amounts of ether, but not without the patients squirming a little. I think some of our bad results are due to the fact that we have given the patients too much of the anesthetic. After the operation the question of nourishment is no more important than that of elimination for the first week or ten days.

XIV.

CAUSATION OF THE SUPPURATION OF THE TEMPORAL BONE.

By William E. Sauer, M. D.,

St. Louis.

The vast majority of suppurations of the temporal bone are secondary to an inflammatory process in some part of the auditory apparatus. In discussing the causation of these suppurations, I will take up the etiology of acute middle ear suppurations, chronic middle ear suppurations, mastoiditis, and the suppurations of the labyrinth.

While it is admitted by some that a purulent exudative process may exist without the presence of pyogenic bacteria or at least without its being possible to demonstrate their presence, it may be safely stated, that practically all suppurative conditions of the temporal bone are due to the invasion and action of pathogenic bacteria. The bacteriology of the subject is to be taken up in the paper that is to follow. Therefore, I will deal only with the mode of invasion and the predisposing causes.

The invasion of the middle ear may take place through the Eustachian tube, through the external auditory canal, by way of the blood or lymph channels, by way of the labyrinth and along the sheaths of the auditory and facial nerves, and from traumatism.

Invasion through the Eustachian tube is the most frequent. Infectious material may be carried by direct extension from continuity of tissue, or may be forced into the tympanum by air or fluids. Inflammatory conditions of the nose and nasopharynx frequently extend through the Eustachian tube into the middle ear by continuity of tissue. This extension of an inflammation or the invasion of the middle ear, is favored by the presence of nasal and nasopharyngeal obstructions with their attendant condition of persistent congestion of the mucous lining of the tube and typanum. These obstructions may not only interfere with the normal ventilation of the tympanic cavity, thus retarding or obstructing the venous circulation,

with resultant exudation into the middle ear cavity. This exudation may become infected, as under these circumstances it offers a favorable medium for the development of pathogenic microorganisms, which, according to Hasslauer, may be present even in the normal middle ear, or which may have gained entrance from the pharynx.

The infection of the middle ear by the air, may take place when attempts are made to clear the nasal passages by forcibly "blowing the nose" when the nose or nasopharynx contains infectious material. This may also occur through the use of the Politzer method of inflation, or when the catheter is employed. Infection through the medium of fluids entering the middle ear through the Eustachian tube, occurs most often from the faulty methods in using the nasal and postnasal douche; through the practice of snuffing up fluids into the nose, or while bathing some of the fluids may accidentally enter the nose and mouth, and find its way into the Eustachian tube. This, I have found to be a rather frequent cause of suppurative otitis media; and in most cases it was determined that the patient had visited one of the indoor swimming pools.

There are a number of acute suppurations of the middle ear which have their origin in the nasal cavities, and which are the result of operative interference. During the period when the electrocautery was so indiscriminately employed in the nose and pharynx, acute middle ear suppurations were frequent sequels; and even to-day this may occur in spite of all precautions. Packing the nasal passages after operative procedures may also be the cause of an acute otitis media; especially if the tampon is allowed to remain in position for too long a time. I have known of a number of instances where an otitis media followed the removal of adenoids and tonsils.

Infection of the middle ear through the external auditory canal, may occur as a result of solution of continuity of the drum membrane, either by traumatism or by the action of an inflammatory process, or through the Rivinian segment. Traumatic perforations, the result of direct or indirect violence, with primary or secondary infection, are not infrequent sources of middle ear suppurations; otitis externa with its extension, according to Dench, through the Rivinian segment, is also a cause of otitis media. More rarely, the development of a vegetable fungus in the auditory canal may lead to a destruction of the drum membrane, with secondary involvement of the mid-

dle ear. The contact of cold water or cold air if continued for a sufficient length of time, may cause an inflammation of the drum membrane with secondary middle ear involvement. Hot water or other irritating fluids poured into the external auditory canal, may produce the same result. Unskilled efforts at removing foreign bodies from the external auditory canal, have resulted in injury to the drum membrane with infection of the middle ear.

Infection of the middle ear by way of the blood and lymph channels, may take place in diphtheria, measles, scarlet fever, tuberculosis, influenza and endocarditis. The infection of the middle ear through the labyrinth or by way of the facial and auditory nerves occurs very rarely, but it may occur in cerebrospinal meningitis or from a meningitis from other causes.

Acute suppurations of the middle ear occur most often in children. The underlying factors accounting for the prevalence of the acute middle ear suppurations in children are, according to Kopetsky:

1. Constitutional diseases, and the hereditary influence of tuberculosis and syphilis.
2. The prevalence of the exanthemata during childhood.
3. Anatomic structures peculiar to childhood in the ear and its adnexa.

Among the general diseases given as predisposing causes are, rachitis, bronchopneumonia, the gastrointestinal derangements, or any disease which may weaken the body resistance. Kopetsky with Koerner holds that predisposing factors are tuberculosis or syphilis in the parent; the children themselves not necessarily being either tuberculous or syphilitic.

Primary tuberculosis of the middle ear is a comparatively rare affection. It is most often encountered as a primary disease in the mastoid. A secondary invasion of the middle ear through the Eustachian tube is not infrequent in older patients, and is seen most frequently in patients with pulmonary tuberculosis; especially during the last stage.

Syphilis, according to Politzer, may also be the cause of a middle ear suppuration; the specific virus entering the middle ear through the Eustachian tube. It occurs most often as a consequence of a syphilitic nasopharyngeal affection which directly involves the tube.

The exanthemata are among the most frequent causes of acute middle ear suppurations, and of these, scarlet fever is the

most frequent and destructive in its effect. According to the views of the majority of investigators, the otitis is most often of a secondary nature, and the infection enters through the Eustachian tube, though it is not impossible that the specific inflammation may involve the pharynx, tube, and tympanum synchronously. In some cases the pharynx is not involved, showing that the infection enters by way of the blood or lymph channels. Levy describes a case of scarlatinal otitis which appeared seven days prior to the eruption. In spite of an early paracentesis the mastoid became involved and operative interference was necessary. The otitis media that occurs in the later stages of scarlet fever runs a much milder course. According to Bezold, one-half per cent of all ear diseases are due to scarlet fever; and in from 20 to 30 per cent of all cases of scarlet fever there is an involvement of the middle ear.

Measles is considered as the next most frequent cause. The characteristic inflammatory macules (in the early stages of the disease) have been found on the tympanic mucous membrane; coincidently with their appearance in the mouth and nasopharynx, and so are not secondary to an inflammatory condition of the nasopharynx. Later in the disease, an infection may take place as the result of an inflammatory process in the nasopharynx.

Diphtheria is also a frequent cause of middle ear suppurations. It rarely occurs as a primary affection of the middle ear. According to Hasslauer, there have been only three bona fide cases reported; namely, those by Krepuskas, Lewin, Gruenert and Schilling. The other cases occur as a secondary involvement through the Eustachian tube.

Influenza is also a frequent cause of acute suppurative otitis media. The infection may enter by way of the blood or lymph channels, and the otitis occur during the first or second day of the disease; or it may occur later, as the result of an infection entering through the Eustachian tube after the disease has practically run its course.

An acute suppurative otitis may also be the result of the direct extension of an inflamed parotid gland, as it occurs in mumps. This extension occurs through the glaserian fissure; or it may occur secondarily from the inflammatory affection of the pharynx and nasopharynx, which sometimes occurs in this disease.

According to Bezold, acute otitis media occurs in from 2 to 4

per cent. of all cases of typhoid fever. The ear involvement occurs in the fourth or fifth week, and may be the result of a pharyngitis or of septic material being blown into the ear in an effort at clearing the nasal passages; or the infection may enter by way of the blood current from an endocarditis or from collections of pus in some other part of the body.

The anatomic structures peculiar to children, which, according to Kopetsky, are factors in tending to cause the prevalence of middle ear suppurations are: the adenoid tissue, the short and wide lumen of the Eustachian tube, and the embryonal character of the contents of the middle ear. He considers that the normal adenoid acts as a barrier to the entrance of particles of food into the Eustachian tube in infants. On the other hand, the effect of the diseased or hypertrophied pharyngeal tonsil with its secretion of ropy mucus and resulting congestion of the pharynx, offers an attractive place for the development of pathogenic microorganisms. Aside from the local effect of obstruction, their influence on the general economy by reducing its resistance, makes the presence of adenoids a most potent factor in the causation of middle ear suppurations.

All writers agree that the widely open lumen of the Eustachian tube, as well as its shortness and horizontal direction as present in children, favors the invasion of infectious material into the middle ear. The retrogressive changes that take place in the embryonal tissue in the middle ear of infants, also furnish, according to Weiss, Preysing, Kopetsky and others, soil for the development of pathogenic bacteria.

Chronic suppurative otitis media is usually regarded as a sequel to acute suppurative otitis media. An exception occurs in tuberculosis; as in this affection the process may appear with a chronic character from the beginning. Just when an acute case passes into a chronic one, is not easy to determine; but after a suppuration has continued for two to three months, it is usually regarded as chronic. The general conditions that influence or cause the transition of an acute into a chronic case are, constitutional diseases which tend to lower the resistance of the patient, such as tuberculosis, syphilis, anemia and marasmus. The conditions which are important local causes of middle ear suppuration are, according to Politzer, 1. The granulations and polypoid growths of the mucous membrane of the tympanic cavity and on the membrana tympani, which develop in the acute stage. 2. The retention and caseation of the puru-

lent secretion in the lateral concavities of the tympanum, in the depression of the petrous bone, and in the cells of the mastoid process. 3. Caries which is developed in the mastoid or in another portion of the temporal bone during the acute course of the disease. 4. Chronic inflammations of the external ear, as is sometimes the case in erysipelas and chronic eczema. 5. Chronic blennorrhea of the mucous membrane of the nasopharynx and ozena.

Just what part the microorganisms play in the development of a chronic suppurative otitis is not definitely known, though an otitis occurring during the course of scarlet fever and diphtheria is more apt to be chronic. There can be no doubt that a number of acute cases become chronic as a result of improper treatment and unhygienic surroundings.

Inflammations of the mastoid process may occur during the course of an acute or chronic middle ear suppuration; or they may be primary and the result of traumatisms. The inflammation of the mastoid occurring during the course of acute chronic middle ear suppurations is due to insufficient drainage, either because of the opening of the drum membrane being inadequate for the accumulated discharge, or from a blocking off of the communication between the mastoid cells and the middle ear. If the views of Politzer are accepted, the mastoid process contains pus in every case of acute suppurative otitis media, and so long as the communication between the antrum and the mastoid cells is free, stagnation of the secretions in the mastoid does not take place, and no reactive symptoms are produced; however, it can be easily understood that if the opening is small, a swelling of the mucous lining of the cells alone may produce an occlusion with stagnation, and as a result, the osseous tissues may become involved. Other causes which tend to mastoid involvement during middle ear suppurations are; exposures to wet and cold, the forcible injections of fluid into the ear, or the employment of the air douche. The experiments of Schutz on the cadaver, have proven that the employment of the air douche in an acute otitis media when the perforation in the drum membrane is small, is a dangerous procedure, as some of the pus may be forced into the mastoid cells. In seventeen of the eighteen cases on which experiments were made, when the perforation in the drum membrane was small, the stained pus placed in the middle ear through this small opening, was found in the remotest cells of the mastoid after catheter inflation, but

he found the pus in only one of sixteen in which the same experiment was made in the presence of a large opening in the drum membrane. The occurrence of mastoid involvement is frequently seen in certain infectious diseases, as in scarlet fever, measles, diphtheria and influenza. The conditions which favor the extension of a chronic middle ear suppuration into the mastoid cells are; narrowing of the external canal, the presence of polypi and granulations and the formation of cholesteatomatous masses, as these interfere with drainage and lead to caries and necrosis of the bone.

Primary inflammations of the mastoid are mostly the result of traumatisms. Tuberculosis is, according to Henrici a comparatively frequent cause in children. He found that one case out of every five mastoid cases operated upon by him, were tubercular; and he believes that the majority were primary. Isemer believes that 13 per cent. of all mastoid cases in children are tubercular, and half of those are primary. Haike demonstrated that out of five cases of tuberculosis of the mastoid examined by him, three were primary.

It is possible that a number of so-called cases of primary mastoiditis are really secondary to a middle ear involvement, so slight as to be overlooked.

The invasion of the labyrinth may take place through the middle ear, by way of the blood and lymph channels, from the cranial cavity by way of the aqueducts, the facial and auditory nerves, and through trauma. The great majority of labyrinth suppurations result from the extension of middle ear suppurations. In the acute otitis media due to scarlet fever, measles, diphtheria and influenza, such extension is more apt to occur. In the chronic cases, tuberculosis and cholesteatoma play an active part. This extension may take place through the anastomosing vessels of the middle ear and labyrinth, or through a destruction of the bone from inflammatory processes. The labyrinth capsule may also be opened during operation on the middle ear, or penetrated by foreign bodies entering through the middle ear. According to Hinsberg, the most frequent paths of entrance for this infection, are the round and oval windows, the promontory and the horizontal semicircular canal.

The invasion of the labyrinth by way of the blood and lymph channels, as it occurs in mumps, and the invasion from the cranial cavity by way of the aqueducts and nerves, as well

as in fractures of the temporal bone, are less important from a surgical point of view.

Regarding the frequency of labyrinth suppurations, Friedrich states that the labyrinth is involved in about 1 per cent of all the cases of middle ear suppurations; but as our knowledge of this subject increases, it will be found that the percentage of labyrinth suppurations will possibly be increased.

Humboldt Building.

XV.

A REPORT OF ATYPICAL AND TYPICAL CASES OF SINUS THROMBOSIS COMPLICATING MASTOIDITIS.

By W. R. Dabney, M. D.,

Marietta, Ohio.

The more cases of sinus thrombosis that I see as a complicating factor in mastoiditis, the more I become convinced that the symptom complex, as laid down in the books,—such as chills, hyperpyrexia, sweating, and a rapid easily compressed pulse,—is not always diagnostic of a septic thrombophlebitis.

There are a number of acute infective conditions that are quite frequently manifested after mastoid operations which so closely resemble sinus thrombosis in their early symptoms, that the diagnosis should be made by exclusion, rather than by the symptoms of sinus infection that may be presented.

Pneumonia is not infrequently seen as a complication after operations upon the temporal bone; that this disease can very closely resemble a sinus infection—in its symptomatology—is well illustrated in the following case:

Case 1.—Child of 12 years. An old chronic otitis media exacerbated by an attack of tonsillitis; operation on tenth day after mastoiditis began. At time of operation the temperature was 99° F. and pulse 100.

Operation.—Subperiosteal abscess over mastoid and temporal fossa; mastoid empyema extending into the superior meatal cells and cells of the anterior wall of the bony canal; perisinuous extradural abscess over the arch joining the horizontal and vertical portions of the lateral sinus.

The sinus was not explored as the history of the case was not very clear, and the patient was not taking the anesthetic kindly.

The posterior bony canal wall was taken down and Panse flaps stitched into the mastoid cavity. The vertical incision over the mastoid was left open.

Six hours after the operation, patient had a chill which

was followed by a rise of temperature to 105° F. and pulse 150. There was frequently repeated slight chills followed by high temperatures and rapid pulse for two days when the characteristic sputa of pneumonia appeared and rendered the diagnosis clear. Auscultation and percussion were negative during this time, as is often found in the early stage of a deep seated central pneumonia.

The pneumonia resolved by lysis, in this case, and the patient was discharged at the end of the sixth week after the mostoid operation.

Acute septic nephritis is another complication that should be considered before it is determined that an infected clot is present in one or more of the venous sinuses of the dura mater.

Case 2.—Purulent mastoiditis in a young man complicated by extradural abscess above the roof of the antrum and tympanum.

Three days after operation, there was a chill, rapid rise of temperature from 99° F. to 104° F., and exhaustive perspiration. The pulse increase was out of ratio to the temperature as it was never above 85 per minute; there was partial suppression of urine and the urinary analysis showed albumin, pus, and cells from the pelvis of the kidney.

The kidney complication cleared up under appropriate treatment.

Case 3.—Child 14 years of age. Acute mastoiditis; ear had been discharging for six weeks.

At the operation, we found only a small quantity of pus and thick bloody serum, in abundance, in all of the cells of the mastoid.

On the sixth day following the operation the patient had a chill which was followed by a rise in temperature and pulse, and there was free perspiration. The next day there was seen a slight croupous exudate on the same side of the throat as the operation. There was tenderness along the upper third of the internal jugular vein, which was no doubt due to the involvement of the anterior cervical lymph nodes that overlie the internal jugular, although there may have been a phlebitis of the jugular by contiguity of structure.

A culture from the throat showed the influenza bacillus.

Under aconite, quinin, camphor and aspirin, the symptoms of sinus thrombophlebitis disappeared.

We have seen a number of cases of gastrointestinal disturbances following mastoid operations, which upon a superficial observation could easily be mistaken for sinus infection, but we have only to make the test for indican to clear up the diagnosis; when it is found, the case is to be treated accordingly and the symptoms of sinus involvement will disappear with surprising rapidity.

Pulmonary, kidney and liver complications as concomitants of an acute osteomyelitis of the temporal bone, are easily accounted for, when we consider the fact there is, in this condition, a thrombosis of the arteries as well as of the veins, and when small particles of these thrombi become detached they are deposited as infected emboli in the above mentioned organs, and thus form a nidus of infection that gives rise to the symptoms which indicate that these organs are involved in a septic process.

CASE 4.—White female, age 14 years. I was called to see this patient on account of an acute exacerbation of a chronic purulent otitis media.

History.—Both ears had been discharging at intervals for ten years. During the apparent quiescent periods, the patient would remove scales of inspissated pus from her ears; has had most of the diseases incident to childhood, and her ear trouble had its inception in an attack of scarlet fever.

Present illness began a week ago during an attack of pharyngitis; ears began to discharge on the second day; the flow from the left ear was very profuse while that from the right ear was scant and foul smelling. During the evening previous to my first visit she had chilling of the lower extremities that was followed by gentle perspiration.

Present Condition.—Temperature, 100.6° F.; pulse, 120. Complains of intense paroxysmal pain in the right ear, the pain being somewhat relieved after there is a free gush of pus; tenderness to pressure applied over the antrum, tip, posterior meatal cells, and mastoid emissary vein; skin over mastoid is glazed and reddened; postauricular groove is obliterated; large perforation in the lower posterior part of drum; decided bulging of inner end of posterior-superior membranous canal.

The streptococcus was the predominating organism in a smear that was taken from the pus as it came through the perforation in the drum.

Operation advised and declined as the father of the child

was out of the city and the consultant did not agree with me as to the advisability of an early operation.

December 14th.—Saw patient at 1 and 9 p. m., and the objective symptoms were more pronounced although she said that she felt better. When operation is proposed it usually has this effect.

December 15th.—Word was received from the father that we operate at once if it was deemed advisable. As the child had had several slight chills during the night and was prevented from sleeping by severe pain in the ear and mastoid, it was thought best that we operate as soon as she could be prepared.

Six p. m. operation; chloroform; the mastoid was densely sclerosed and not a single cell was encountered until the antrum was reached when pus and granulations as well as cholestoatoma were encountered; antrum was larger than usual and the walls were undergoing ulcerative degeneration; the posterior canal wall was chiseled away and the malleus and incus, which to the unaided eye appeared to be normal, were found imbedded in a mass of fibrous connective tissues that had formed between the drum and the inner tympanic wall; this condition in connection with the granulation formation in the antrotympanic duct prevented free drainage from the antrum into the middle ear and accounted for the paroxysmal pain as the antrum was not emptied until the pressure became great enough to force the pus into the tympanic cavity. This is a diagnostic point of some importance as in my limited experience I have always found pus in the antrum when there was repeated attacks of paroxysmal pain that were relieved by a free flow of pus through a large perforation in the drum membrane.

The eburnating process had not advanced so much in the lower two-thirds of the mastoid but the cells of this region were small and filled with cholesteatoma, constituting a cholesteatomatous infiltration of the mastoid—a rare condition in my experience as this is the second case that I have met with in my practice.

There was a small perforation in the inner table overlying the lower portion of the sigmoid sinus, from which pus was bubbling; perforation was enlarged and a dram of pus liberated. The sinus wall was beginning to soften, and as palpation did not afford us any information, the outer wall was in-

cised between two pressure pads and a fair sized parietal thrombus was taken out of the sinus.

This patient had a slow and tedious recovery, due to the lighting up of an old nephritis.

This case very forcibly illustrates the fact that we may have an infected thrombus, in one of the sinus of the dura mater, and yet there may not be manifested the symptom complex that we usually expect to see in these cases.

CASE 5.—This case came under my observation in 1904, at the instance of Dr. E. W. Hill.

History.—White male, age 23 years. Frequent attacks of earache in the left ear, but there has never been a noticeable fluid discharge from the ear, although he has frequently taken greenish crusts of a pungent odor from this ear. Present trouble began three weeks ago during an attack of tonsillitis, but mastoid swelling did not develop until two weeks later, that is one week previous to his first visit to my office.

Present Condition.—Temperature, 98° F.; pulse, 96. Pain deep down in the ear; edema limited to the lower two-thirds of the mastoid. Slight pressure over tip and antrum caused intense pain. Ear was freed of inspissated pus and cerumen. Inner end of membranous canal was found to be sagging and boggy. There was a large sequestrum of bone in the fundus of the canal, which could not be delivered through the external meatus. Mastoid does not transilluminate.

Saw patient at 3 p. m. and his temperature was 100° F., pulse, 100. He has had a number of attacks of vertigo and several rigors since his morning visit. The hearing power, for both high and low tones, is very much impaired. Advised operation at once, but the young man was under legal age and the consent of his parents could not be obtained before the following day.

June 10th.—Temperature, 98.8° F.; pulse, 98. Chill during the night which was followed by free perspiration. The ear is discharging a thin blood streaked material, from which a smear was taken, and the streptococcus was found to be the predenominating organism.

Four p. m.—Temperature 100.5° F.; pulse, 115. Marked increase in the objective symptoms. The father tells me that the boy had an acute ear condition in early infancy and that since that time there had been an accumulation of greenish malodorous crusts about the external meatus.

June 11th.—Operation; chloroform; subcortical cells and a large tip cell contained pus; antrum and tympanic cavity were filled with pus and cholesteatoma. The antral walls were undergoing dry caries as the bone came away under the curette like grains of sand.

A sequestrum comprising the inner end of the posterior and inferior walls of the external auditory canal was taken out, thus leaving the facial nerve exposed in its vertical portion. The inferior and anterior walls of the tympanic cavity were absent and the jugular bulb, the internal carotid artery, and the capsular ligament of the glenoid fossa were exposed. The wound was packed with strips of iodoform gauze and left open.

The young man was relieved of his pain and vertigo by the operation, but the fluctuations of temperature and pulse, and the slight chills were repeated three and four times a day; each chill was followed by a rise of temperature and pulse and mild perspiration. This condition continued for ten days before the consent of the parents could be obtained for another operation. During this ten days the temperature varied from 97° F. to 101° F. and the pulse from 110 to 140.

As the mastoid wound was in good condition, and the jugular bulb previous to the operation had been in gross communication with the pyogenic material in the tympanic cavity, it was thought that we had to deal with a primary thrombosis of the jugular bulb, and that small particles of the infected clot were becoming detached into the general circulation and thereby causing the form of bacteremia that was manifested.

On the tenth day following the primary operation the patient was anesthetized and the sigmoid sinus uncovered down to the bulb. A palpable clot was not discoverable, therefore, an aspirating needle of large caliber was inserted into the sinus and a free flow of blood followed but, when pressure was applied to the sinus above the needle, the flow immediately ceased. Pressure was maintained and the portion of the sinus was laid open with a small knife; a curette was introduced, and particles of softened clot were removed but we were unable to re-establish the circulation. It was deemed best to resect the jugular before resorting to further manipulations to free the sinus of the thrombus.

After the vein was excised, the sinus was incised down to the bulb and a great deal of disintegrated clot was taken out.

A gauze drain was inserted upward into the bulb, in order that it might bring away any softened material that had escaped our efforts. The temperature and pulse became normal within four days after the sinus operation and remained so throughout his convalescence.

This case serves to illustrate the difference between an actual involvement of the labyrinth by the pyogenic process and a labyrinth disturbance due to pressure transmitted through the fenestra in the inner tympanic wall, as the labyrinthine symptoms disappeared as soon as the pressure was removed by operation.

The low form of bacteremia that was manifested in this case may be accounted for in several ways: (1) That the clot in the bulb and lower part of the sigmoid sinus was disintegrating slowly, and thus such small infected emboli were detached into the blood stream that the phagocytes were able to perform their function as scavengers before the extreme constitutional disturbances, that are usually seen in a bacteremia, could be manifested. (2) It is quite possible that here may have been an attenuation of the organism responsible for the infection in consequence of the long continued course of the suppuration, just as we see it in chronic gonorrhea. (3) The individual equation of susceptibility and immunity or resistance to infection must be considered in a given case as the same conditions of infection in an individual of good resisting powers would not produce the violent train of symptoms that would be manifested in an individual that was greatly debilitated before the infection.

The wound in the neck healed by primary intention, and this, I think, was due to the method of technic that was followed in the resection of the vein. The technic of the procedure was as follows: A heavy pad of gauze was passed under the vein, preliminary to cutting it between the double ligatures, in order to prevent septic material in the severed ends of the vein from infecting the wound in the neck.

Before the stumps of the severed vein were dropped into the neck, their intimas were cauterized with carbolic acid and alcohol, just as the general surgeon does the stump of the severed appendix before he returns it to the abdominal cavity.

I have followed the above technic in several jugular resections, with the most gratifying results, as to primary union.

of the cervical wound, in cases that were mani
degree of bacteremia.

CASE 6.—White male, age 16 years. I saw this case in consultation with Dr. W. H. Metcalf, at 2:30 p. m., September 5, 1907.

History.—Discharge from left ear since early childhood as sequel of scarlet fever. There had been pain back of the ear on numerous occasions, when the discharge from the ear would cease temporarily.

Present trouble began about ten days ago during an attack of tonsillitis. He has had a number of mild chills and his temperature has fluctuated between 100° F. and 102° F. He had a very hard chill this morning, but his temperature was not taken until 1 p. m. when it registered 101° F.

Present Condition.—Temperature, 102.6° F.; pulse, 130; respiration, 22. He has an anxious drawn facial expression, that is suggestive of fear; skin is of a muddy yellow color; tongue is heavily coated; breath is foul; speech is slow and voice tremulous; there is an annoying dry cough but ascultation and percussion are negative. Has constant pain over entire left side of the head but is most severe in the mastoid region; he is extremely sensitive to pressure lightly applied over the tip, antrum, emissary vein and along the course of the internal jugular vein. There is edema, beginning about two inches above the external auditory meatus and extending downward to the clavicle. This edema is particularly noticeable in the occipital triangle, and there is exquisite tenderness to pressure applied in this region.

External canal is full of thick foul pus; drum membrane, malleus and incus are missing, and the inner tympanic wall is covered with polypoid granulations. The streptococcus was found to be the predominating organism.

There is a slight Babinski reflex and Kernig's sign is fairly well marked, as the angle of extension is about 120°; vision is indistinct and ophthalmoscopic examination revealed a double optic neuritis. The patient readily recognizes articles handed him and understands the purposes for which they should be used, but he displays considerable difficulty in naming the articles submitted to him.

There is a stable mydriasis of the left pupil and the right one responds very sluggishly.

Diagnosis.—Mastoid empyema, sinus thrombosis, and a pro-

visional diagnosis of meningitis with brain pressure due to an extradural or subdural pus collection.

Admitted to hospital at 4 p. m.; temperature, 101.6° F.; pulse, 116; chill at 6 p. m., lasting 15 minutes; 7 p. m. temperature, 105.5° F.; pulse, 140; respiration, 28.

Operation began at 10 p. m. and the temperature was 102° F. The usual mastoid incision was later supplemented by posterior incision extending to the inion. Opening the cortex set free a thick, dark offensive fluid that was under considerable pressure as numerous bubbles could be seen in the fluid as it gushed out; large antrum was filled with cholesteatoma; inner table overlying the vertical limb of the sinus was completely eroded and the outer wall of the sinus was covered with granulations from which pus was exuding; granulations were removed and a perforation was found in the wall of the sinus; enlarged perforation and a disintegrated clot were mopped out of the sinus; ulceration of visceral wall of the sinus at the knee; ulcerated part of the sinus wall was cut out with small scissors and about three drams of pus was evacuated, exposing an area of superficial cerebral necrosis at this point. This is about the region of the naming center as pointed out by Mills, and possibly accounts for the patient's hesitation in naming objects.

On account of the extensive softening of the clot in the sinus, it was thought best to ligate and excise the internal jugular vein before attempting to free the sinus of the disintegrated thrombus. The jugular was found to be thrombosed to one-half inch below the facial vein, the latter vein also contained a clot. The jugular was excised below the lower limit of the clot and the stump of the vessel was stitched into the upper angle of the cervical incision.

Sinus had to be uncovered to the torcular before the posterior axis of the softened thrombus could be reached. This sinus was next curetted in the direction of the bulb but a return circulation was not established. A stream of normal saline solution was introduced into the sigmoid sinus in the direction of the jugular bulb and particles of softened clot washed out of the end of the jugular that was stitched into the neck wound. The superior petrosal sinus was opened inward as far as the internal auditory meatus without establishing a return flow.

The posterior osseous canal wall was taken down and the

middle ear freed of granulations. Posterior horizontal incision closed and wound packed with gauze. Lower two-thirds of neck wound closed but it suppurated and had to be reopened.

September 6th, 6 p. m.—Temperature, 99.4° F.; pulse, 84. Chill at 6:30 p. m., chill at 10:30 p. m., following which the temperature was 104.2° F. and pulse 130.

September 7th.—Chill at 8:30 a. m., followed by a rise of temperature to 104° F. and pulse 140. During the night he complained of pain over the upper portion of the left lung; the respirations are shallow and increased in frequency; large moist rales heard on auscultation and there is prune juice expectoration; has developed a diarrhea and the stools are extremely offensive.

Dressing changed and the entire wound found covered with pyogenic membrane; all stitches taken out and wound swabbed with turpentine; hot compresses of permanganate of potash solution ordered which were to be changed every twenty minutes. Chill at 7:30 p. m.; one hour afterward the temperature was 103 6° F., pulse 160.

September 8th, 7 a. m.—Temperature, 99.5° F.; pulse, 96. Chill of thirty minutes' duration and the temperature soon registered 104.5° and pulse 126. Slight edema and discoloration of the lids of the left eye.

September 11th.—For the past forty-eight hours the temperature has fluctuated between 98° F and 102° F., and the pulse has varied from 96 to 116. Patient has been exceedingly restless and at times wildly delirious; Kernig's and Babinski's signs are positive and there is some retraction of the head; edema and discoloration of the lids of the left eye have increased.

September 12th.—Exophthalmos of the left eye; edema of lids of right eye; projectile vomiting several times during the day; unconscious at 9 p. m. and died six hours later.

Autopsy not permitted.

This patient was given 140 cc. of streptolytic serum and twelve hypodermic injections of Declat's preparation of phenic acid besides the usual remedies that are in vogue in the treatment of pneumonia.

This case as well as the preceding one serves to illustrate the fact that in an acute exacerbation of a chronic purulent otitis media in which there are symptoms of mastoiditis, we should resort to radical operative interference at once if we

wish to conserve the interests of our patient. They also serve to show us the extensive destruction that may take place in the temporal bone without the manifestation of subjective symptoms. From the findings at the operations upon these patients I am of the opinion that chronic suppuration of bone had been in progress for months, if not years, as it is hardly conceivable that such destruction of osseous tissue could be accomplished in one or two weeks. Every surgeon who has had a wide experience in dealing with chronic suppuration of bone, has often been surprised at the mildness of the symptoms and the advanced pathologic conditions that he finds at the time of operation.

On the day following the operation, the young man appeared to be better, inasmuch as the pain was relieved, and he was able to name objects readily, the later condition would indicate that the naming center had not been destroyed, but as has been suggested by Mr. Ballance, the connecting fibres between this center and the speech center were obtunded to a certain extent by pressure from the subdural abscess.

Cases, like the one under discussion, which manifest symptoms of meningitis before operation, do not offer a very favorable prognosis, and in this case the Babinski and the Kernig signs were positive, and we were fully warranted in assuming that we had a complicating meningitis.

As this patient had an annoying cough previous to the operation, although auscultation and percussion were negative, I cannot but conclude that a metastatic pneumonia was developing at that time, as clinicians of experience in diagnosticating diseases of the lungs tell us that it is often impossible to make a diagnosis of central pneumonia by auscultation and percussion, and that they have to wait for the appearance of the characteristic expectoration.

I am well aware that, during the manipulations that are necessary to clear a thrombosed sinus, one may dislodge a portion of the infected clot into the general circulation, and that it may be carried by the blood stream to the right heart, and thence to the lungs, to form the focus for a metastatic pneumonia, but I cannot concede that this occurred in the case before us, as the clot in the bulbar end of the sinus was not disturbed until after the internal jugular had been excised, and pressure was made on the opposite jugular while we were clearing the torcular end of the sinus.

The foul smelling purulent stools that the boy had would indicate that his diarrhea was due to a septic enteritis and not to an excess of carbon dioxid in the blood which was the cause of the frequent loose stools in the late stage of pneumonia.

The clinical symptoms of exophthalmos, edema and discoloration of the lids of the left eye, and the later manifestation of the same symptom in the right eye in connection with the fact that we were unable to get a return flow from the superior petrosal sinus, would amply justify us in concluding that there was a septic thrombophlebitis of both cavernous sinuses; this being the case it is quite easy for us to see how the infecting element could be carried to the lungs through the right internal jugular.

It is in cases like this one that the diagnosis of a complicating brain abscess is rendered extremely difficult, if not impossible, as the symptoms of pyemia so predominate that they obscure the characteristic signs of low temperature and pulse that are seen in abscess of the brain.

XVI.

DIAGNOSIS AND TREATMENT OF SUPPURATION OF THE LABYRINTH.

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The diagnosis of suppuration of the labyrinth is made from the symptoms evinced by the patient before operation and by the pathologic condition discovered during operation.

Symptoms.—The invasion of the labyrinth by a suppurative process in the middle ear is marked by no symptoms in the majority of cases. This is due to the fact that it occurs generally during a chronic suppurative otitis media associated with caries or cholesteatoma. Extension takes place slowly by a process of erosion from without. This allows the labyrinth apparatus to accommodate itself to the gradual change in pressure and condition that occurs with the localized destruction of its bony capsule. It is the sudden invasion of the labyrinth or the sudden extension of disease within a labyrinth already entered that gives rise to symptoms. When this occurs it causes certain manifestations that are of diagnostic value.

Of these deafness is of importance. Unilateral deafness, as Bezold has pointed out, is difficult of diagnosis and consequently of doubtful value. When, however, a patient suffering from a suppurative otitis media develops under observation a sudden increase in deafness in the suspected ear, the symptom is of great significance. The absence of deafness does not necessarily mean that extensive pathologic change has not already taken place in the labyrinth for the vestibule may be filled with cholesteatoma and pus without materially affecting the hearing. Even the cochlea may be extensively diseased without destroying hearing for conversation.

Disturbances of equilibrium are frequently present. When standing the patient often rests most of his weight upon the foot of the affected side. While walking he tends to turn off or bend to the affected side.

These disturbances are intensified if the eyes are closed. In some cases they are slight but continuous while in others they alternate with periods during which the patient is free of all inconvenience. In severe cases they are so marked as to prevent the patients from assuming the upright position or moving about without support. After being present for some length of time they have been known to disappear for years.

Vertigo is common and is intensified by exertion. It varies greatly in degree and character. In some cases it is not more than dizziness, transient or continuous. In others typical and severe rotary vertigo appears. In one of my cases pressure upon the right semicircular canal after operation caused all objects to rotate very rapidly to the right. Removal of pressure causes them all to snap back through the reverse arc quickly until they reached their proper position. Pressure upon the posterior semicircular canal caused the bed to slip suddenly away toward the foot. Removal of pressure brought it quickly back again. These sensations were so intense that that patient would hold tightly to the bed to keep from throwing himself on to the floor.

In cases in which vertigo is most marked nausea and vomiting are generally noted. They occur upon arising or follow exertion.

Lateral nystagmus is less common. It rarely occurs without vertigo and is most marked when the eyes are turned toward the healthy side.

Fever and headache are rather uncommon and are not at all characteristic.

The hearing test is of questionable value. The hearing for the voice in my own cases varied from the whisper at three feet, to a degree which made it probable that the loud conversational voice was heard in the other ear although that ear was hermetically sealed. The fact that a very high degree of deafness often occurs in chronic suppurative otitis media without extension of the infection to the labyrinth and that extension into the semicircular canal system sometimes takes place without causing marked deafness, lowers the value of the voice test considerably even when taken into consideration with other tests and renders it worthless when considered alone.

The Weber is decidedly unreliable. It was not localized in several of my cases and in others was localized in the suspected ear quite as frequently as in the other.

The Rinne was negative more often than it was positive.

The high limit, however, was always somewhat lowered, in one case was 512. In this case the Weber was not localized. The lower limit is of course elevated on account of the middle ear involvement.

The symptoms mentioned are important in that they point toward the presence of an intracranial complication but are not sufficient in themselves to render a diagnosis of labyrinth disease positive. They may all be present in cases in which the labyrinth is not involved. This is demonstrated by the following case:

Mrs. S., 30 years. Scarlet fever at the age of 4 years. Both ears discharged at that time. Right ear soon ceased to discharge but left has continued to discharge at times ever since. At time of first discharge had complete left facial paralysis. This lasted for about two months. Since then has had times when paralysis was apparent lasting several weeks. Since first attack has never been complete. About two weeks ago during a head cold was taken with severe rotary vertigo, ringing tinnitus and increase in deafness in the left ear, pain in the left occipital region, slight foul discharge from left ear. Temperature has reached as high as 105 with frequent remissions; no chills, yesterday delirium, pupils dilated, breathing difficult, apparently in extremis. Today remarkable change for the better.

Examination.—Patient well developed, intelligent woman, perfectly rational, but inattentive, complaining of pain in region of left ear, high pitched tinnitus left ear, temperature 101, pulse 80, respiration 20, leucocytosis 14,000. Knee jerks slightly exaggerated. Right ear shows evidence of old suppuration but no sign of acute inflammation. Left ear, membrana tympani present, retracted, scar in superior half, small perforation in postero-superior quadrant, contains somewhat tenacious pus, probe passes into attic. No drooping of posterior superior canal wall, no mastoid tenderness. Hearing test somewhat unsatisfactory on account of patient's condition.

Right.		Left.
Loud conversation	Voice.	Loud conversation
Not localized.	Weber.	Not localized
Negative	Rinne.	Negative
256 slightly higher forks 0	A. C. . .	256 slightly higher forks 0
Apparently somewhat decreased	B. C. . .	Apparently somewhat decreased.
Dench 3.	High.	Dench 4
256.	Low.	256

Paracentesis permitted escape of foul pus and cholesteatoma. Diagnosis of chronic suppurative otitis media, cholesteatoma and probable involvement of the labyrinth was made and radical mastoid operation advised with exploration of labyrinth if indicated at time of operation. Operation was refused. Patient remained in same condition for two days and then had another attack of pain.

Rise of temperature to 105 without chill, increase in deafness, tinnitus and vertigo from which she made a slow recovery. Ten months later a radical mastoid operation was performed. Tympanum and mastoid were found filled with cholesteatoma but the labyrinth was intact. Complete recovery. It is interesting to note that after the radical operation the upper limit became normal and hearing for conversational voice increased to six feet.

The labyrinth symptoms and hearing test indicative of labyrinth involvement were no doubt due to pressure upon the labyrinth of the cholesteatoma and to incident congestion.

Even more important than the fact that symptoms indicative of invasion of the labyrinth may be present during the course of a chronic suppurative otitis media without disease of the labyrinth is the fact that invasion of the labyrinth may take place without the presence of a single symptom on account of which the condition may be suspected. Of my own cases marked deafness was present in three, disturbances of equilibrium in five, vertigo in five, nausea and vomiting in three, nystagmus in two, no symptoms whatever in five.

The diagnosis of labyrinth suppuration can at present be made with certainty, in the great majority of cases at least, only at the time of the radical mastoid operation, by discovery of disease of the inner tympanic or mastoid wall and of a sinus leading into the interior of the labyrinth. These sinuses appear in order of their frequency in the semicircular canals,

through the oval window, through the promontory, and through the round window. Perforations into the horizontal semicircular canal is frequently easy of diagnosis. This is true when the perforation occupies a point just below the most prominent portion of the semicircular canal. In such cases it appears as a single opening into the lumen of the canal, or if more destruction has taken place as two openings one leading into each branch of the canal. (I have seen several such perforations without signs of caries, granulation tissue or pus within the canal.) The diagnosis is more difficult although the destruction is greater in cases in which the posterior half of the horizontal semicircular canal is lacking. With the perforation in this position, the operator may be left in doubt as to its existence despite perfect illumination and painstaking investigation. Pus is rarely seen issuing from such a perforation.

I have seen it in but one case. In that case the vestibule was filled with fluid pus. More frequently a small bud of granulation tissue is noted within the canal. When this is wiped away the observer may be in doubt as to whether the interior of the canal is involved or not, possibly concluding that the granulation tissue is due entirely to the disease within the antrum. I believe that such indecision is justified and that perforation may occur in the semicircular canal, exist for a time, become closed by granulation tissue and later by bone with but slight, if any, involvement of the labyrinth. In one case, in shaving down some cancellous bone over the horizontal semicircular canal in a patient in whom no labyrinth symptoms had been noted I cut away a small irregular mass of bone, the removal of which opened the canal. This I think was new bone formed in the successful attempt on the part of nature to close a perforation and protect a labyrinth from invasion.

The posterior semicircular canal is somewhat more deeply situated than the horizontal and is more deeply covered with cancellous bone consequently its exact delineation is somewhat more difficult. On this account perforation through the canal is sometimes missed. When the canal is thoroughly exposed the perforation is easily seen occupying the most prominent portion of the canal. It is sometimes seen on the anterior surface and associated with destruction of the posterior branch of the horizontal canal. In some cases very small pneumatic cells may cover the canal and be especially

numerous in the neighborhood of the solid angle. They sometimes extend rather deeply along the walls of the canals and if filled with freely bleeding granulation may be mistaken for a perforation. In this connection the systematic use of the fine probe is of great diagnostic value. This instrument must be employed with judgment. It is well recognized that in cases which exhibit a single perforation in this neighborhood the labyrinth disease is frequently definitely localized and that this localizing process takes place by the formation of a wall of protecting granulation at the deepest limit of the disease. It is without question highly dangerous to pass a probe further into the semicircular canal than this protecting wall. Such a procedure only carries infection into the deeper portions of the labyrinth and disseminates the disease. It does not afford the observer any additional information. As the point at which this protecting wall has formed can not be told, the probe is to be passed only deep enough to render the diagnosis of perforation positive.

It is generally stated that next in frequency to the neighborhood of the semicircular canals perforation takes place through the oval window. In the presence of the stapes this perforation must be difficult of diagnosis. In the absence of the stapes, perforation can sometimes be made out if not by inspection, then by means of the fine probe. Hinsberg states that infection can pass through the oval window in cases in which macroscopic change in the window cannot be discovered.

In support of this statement I have been able to demonstrate caries of the vestibule in a case in which I could not discover a perforation. The absence of the stapes is not always significant of perforation through the oval window as it was noted in one of my cases in which no perforation could be detected and in which fair conversational voice was present after operation. I have seen perforation of the promontory in one case in which about two-thirds of the promontory was lacking. Cases have been reported in which infection reached the labyrinth from the cranial cavity through the internal auditory meatus or the aqueducts. I am inclined to think that the acqueductus vestibuli was the avenue of infection in one of my cases.

Much difference of opinion exists concerning the pathologic

significance of perforations in various positions in the external labyrinthine wall.

In my cases, judging from hearing test and result of operation, single perforations into the horizontal semicircular canal occurred only in cases of circumscribed labyrinthine disease. Double perforations, one in the horizontal and one in the posterior semicircular canal, occurred in two cases in which the vestibule was filled with pus and cholesteatoma. It would seem that double or multiple perforations must always speak for an extensive pathologic condition. In considering the value of visable perforations in the diagnosis of labyrinth suppuration secondary to suppurative otitis media it must be remembered that the disease has been demonstrated clinically and at autopsy in cases in which perforation could not be recognized.

The indications for operation upon the labyrinth are met in two classes of cases:

1. Those in which symptoms referable to labyrinth disease have not been present before operation.

(a) When a single perforation is discovered through a semicircular canal, from which pus escapes.

(b) When a perforation through the oval window is discovered, whether pus is seen or not. In these cases marked deafness is present and extensive disease of the vestibule must be suspected.

(c) When labyrinth symptoms develop suddenly after a radical mastoid operation. The majority of these cases are due to a latent labyrinth suppuration, an acute exacerbation of which has been set up by the operation. In such cases the vestibule is to be opened widely and further operative measures governed by its condition. In isolated perforations through the horizontal semicircular canal in which no other evidence of disease can be demonstrated operation upon the labyrinth may be postponed.

This is in support of Hinsberg's statement that in his experience, of all cases of circumscribed semicircular canal disease not a single patient succumbed to the consequences of the labyrinth suppuration. In this connection he points out the necessity for watching, very carefully, cases in which a single perforation of a semicircular canal has been demonstrated so that on the appearance of vertigo, nystagmus and diminution of hearing, the labyrinth may be promptly opened.

2. Those in which labyrinth symptoms have been present before operation:

(a) When no fistula is found.

(b) When fistula is found.

When no fistula is found. This class of cases, it seems to me, should be very small and should include those cases which have developed labyrinth symptoms while under observation and those in which deafness typical of disease of the perceptive apparatus can be demonstrated. Symptoms highly suggestive of labyrinth suppuration so frequently disappear after the radical mastoid operation that unless the pathologic change in the tympanum and mastoid is altogether too slight to account for the labyrinth symptoms delay may be allowed. To this class belong also those cases in which a deep seated cerebellar abscess or extradural abscess is suspected (Jansen).

When a perforation into the labyrinth is found in a case that has previously shown labyrinth symptoms the indication for opening the labyrinth is clear.

The following six cases are illustrative of the fact that invasion of the labyrinth can take place without symptoms, while the next five show some symptoms characteristic of the condition.

August 17, 1905. CASE 1.—Percy B., 15 years. Discharge from left ear following scarlet fever eleven years ago. Discharge has persisted with infrequent remissions ever since. No symptoms except moderate deafness and foul discharge.

Examination.—Right ear negative. Left, great destruction of M. T. Tympanum filled with granulation, pus and cholesteatoma. No mastoid symptoms. Large amount of adenoids.

Right.	Hearing Test.	Left.
Loud conversational voice..	Voice	20/21
+	Weber	.....
—	Rinne	+
Dench 3	High	N.
512	Low	N.

Radical mastoid operation discovered tympanum filled with cholesteatoma, antrum containing moderate amount of granulation tissue, pus and cholesteatoma, malleus and incus wanting. Small sinus leading into posterior branch of horizontal semicircular canal. No evidence of pus in canal, no caries. Koerner flap and skin grafts later. Sinus moderately en-

larged for purpose of examination. Adenoid operation. Slight vertigo after operation.

May 3, 1906. CASE 2.—Ivan P., age 10 years. Chronic suppurative otitis media since scarlet fever seven years ago. No symptoms except moderate deafness and discharge.

Examination.—Right ear. Great destruction of membrana tympani. Ossicles not seen. Tympanum contains granulation, pus and cholesteatoma. Probe passes into attic and discovers caries on external attic wall and in direction of antrum. Left ear: Scars in membrana tympani. No evidence of present suppuration.

Right.	Hearing Test.	Left.
L. C. V. at ear	Voice	15/21
+	Weber	
—	Rinne	+
Dench 2	High	N.
512	Low	256

Right radical mastoid operation. Cholesteatoma in tympanum, antrum and perisinus collection of cholesteatoma. Perforation in horizontal semicircular canal leads into vestibule. This opening enlarged. Thiersch grafts in Eustachian tube and tympanum. Koerner flap. Initial incision sutured. Recovery without marked labyrinth symptoms.

July 8, 1907. CASE 3.—Fred H., age 17 years. Discharge from right ear for fifteen years without symptoms.

Examination.—Right ear: Polyp occludes canal, from around polyp, escapes foul cholesteatoma. Sinus through posterior canal wall passes into mastoid.

Right.	Hearing Test.	Left.
Loud conv. voice	Voice	20/21
+	Weber	
—	Rinne	+
Dench 2	High	N.
512	Low	N.

Radical mastoid operation: Tympanum, antrum and mastoid filled with true cholesteatoma. Dura over middle cerebral fossa and over sinus laid bare by the disease. Polyp attached to inner wall of antrum. Sinus through horizontal semicircular canal leads into vestibule. Sinus enlarged. Koerner flap, initial incision sutured. Moderate vertigo for several days after operation. Discharged well.

November 21, 1907. CASE 4.—Madge S., age 8 years. Recurrent discharge for the past three years, continuous from the left ear for the past year. No history of labyrinth symptoms.

Examination.—Membrana tympani shows great destruction. Tympanum contains cholesteatoma and pus. Ossicles not visible. No mastoid tenderness. After several weeks' treatment without avail, radical mastoid operation. Most of the disease in and in the vicinity of the antrum, which contained cholesteatoma and caries. Fallopian canal lacking for about one-fourth inch in the immediate vicinity of the horizontal semicircular canal. Horizontal semicircular canal shows almost complete destruction; remainder of canal carious. Dura and sinus laid bare. Short Koerner flap. Initial incision sutured. Skin graft later. Complete recovery without labyrinth symptoms.

December 30, 1905. CASE 5.—Frances McD., age 18 years. Continuous discharge from both ears since measles thirteen years ago. Severe headache but no other symptoms.

Examination.—Both ears showed marked destruction of membrana tympani. Tympanum contains cholesteatoma and pus. Caries in both attics. Left shows considerable destruction of posterior canal wall.

Right.	Hearing Test.	Left.
8/35	Voice	5/35
Dench 2	Weber	+
—	Rinne	—
	High	Dench 2
512	Low	512

January 11, 1906.—Radical mastoid operation left side. Extensive sclerosis. All of posterior and posterior half of horizontal semicircular canal found destroyed. Opening enlarged. Koerner flap. Thiersch grafts to tube and tympanum. Moderate vertigo after operation.

Stacke operation on the right side at a later date. Labyrinth not involved.

August 28, 1905. CASE 6.—Cleo F., age 15 years. Both ears have discharged since the age of 6 months. No nausea; no dizziness or other labyrinth symptoms.

Examination.—Both ears show canals filled with granulations, pus and cholesteatoma. Sinus leads through the pos-

terior canal wall of the left ear into mastoid. Adenoids and tonsils.

Right.	Hearing Test.	Left.
L. C. V.	Voice	L. C. V.
Dench 3	Weber	+
—	Rinne	—
	High	Dench 3
1024	Low	1024

Radical mastoid operations on both ears at different dates. Right side showed moderate amount of cholesteatoma, granulation tissue, and pus throughout tympanum and mastoid. Labyrinth had been invaded through the posterior semicircular canal which had been completely destroyed. Horizontal semicircular canal also involved. Thiersch graft of tympanum, initial incision sutured. Moderate labyrinth symptoms after operation. Mastoid operation followed by removal of adenoid.

Left radical mastoid operation showed extensive cholesteatoma formation with exposure of the facial nerve but no involvement of the labyrinth.

March 15, 1907. CASE 7.—Frank R., age 14 years. Right ear has discharged for ten years, cause unknown. No symptoms until one month ago when complete facial paralysis and vertigo with increase in deafness. Patient says that at that time and for several days, he staggered to right but did not fall.

Examination.—Canal occluded in its middle third by drooping of superior canal wall which touches inferior. By inserting speculum beyond occluded area small amount of stale discharge and glistening polyp are seen. Complete facial paralysis. No mastoid tenderness. Adenoids and tonsils.

Radical mastoid operation. Tympanum, antrum and mastoid filled with foul pus. Fallopian canal shows perforation about one-fourth inch long, downward from level of horizontal semicircular canal which shows perforation, which uncaps canal and shows openings into both branches. Openings enlarged. Koerner flap. Initial incision sutured. Insignificant vertigo after operation. Adenoids and tonsil operation at a later date. Complete recovery from facial paralysis in three months.

May 13, 1906. CASE 8.—Wm. H., age 22 years. Right ear has discharged since measles seventeen years ago with occasional exacerbations. Polyps removed from both ears one

year ago. Two months go while undressing had his first attack of vertigo which was so severe that he fell over on to the bed. This vertigo has continued with periods of remission and exacerbation up to the present time. No nausea or vomiting. Acknowledges syphilis several years ago. No recent symptoms.

Examination.—Patient sits with head bent to right. Not interested in surroundings. Answers questions slowly but correctly. Apparently not in pain. Pupils equal, react normally, no nystagmus, no strabmisus. Disturbance in equilibrium marked upon arising, stands with most of his weight upon the right foot, while walking turns off and bends to the right.

Ears: Right, canal contains creamy pus. Depths of canal filled with easily bleeding granulations. Drooping of superior wall of the canal just external to the tympanum. No mastoid tenderness. Left, dry depths are filled with bloody crusts.

Right.	Hearing Test.	Left.
L. C. V.	Voice	2/35
Not localized	Weber	Not localized
—	Rinne	—
512	High	N.
512	Low	512

Radical mastoid operation right side. Marked superficial sclerosis of mastoid. Deeper parts filled with cholesteatoma. Horizontal semicircular canal completely destroyed. Granulations in tympanum. Koerner flap, sewed to posterior wall of concha. Initial incision not sutured. Riverdan skin grafts at a later date. For several days severe rotary vertigo to the right. This gradually wore off. While walking the patient had the inclination to turn off to the right but was able to walk in a straight line with some effort. Three months after operation had two attacks of severe vertigo with some nausea. Examination revealed complete epidermitization of the operative field. No apparent trouble. Since then no report of patient.

August 5, 1905. Case 9.—Patty Van T., age 9 years. Has had continued discharge from both ears since scarlet fever six years ago. Two weeks ago severe pain right side, with decrease in amount of discharge, postaural swelling on the third day. Since attack patient has been very dizzy but has been too sick to complain.

Examination.—Patient evidently very ill. Temperature 103, pulse 110, very weak and thready, sepsis, adenoids.

Right ear, canal occluded by drooping of superior wall, large postaural swelling extending to cheek. Cholesteatoma in canal.

Left ear, membrana tympani gone, tympanum filled with cholesteatoma. Right radical operation with curettage of vestibule. Cortex, paper thin. Five perforations externally. Tympanum, antrum and mastoid filled with cholesteatoma. Two perforations in middle cerebral fossa. Pus and cholesteatoma on sigmoid sinus. Posterior semicircular canal and posterior half of horizontal semicircular canal destroyed. Vestibule filled with pus. Left radical mastoid at later day. Labyrinth not involved. Discharged well.

October 8, 1906. Mrs. Wm. J. B., age 42 years. Patient had scarlet fever at age of 5 years. Both ears discharged for several years thereafter. Later the right ceased but the left has troubled her until the present time, slight facial paralysis. For about a year she has had frequent and severe sick headaches, with nausea, vomiting and rotary vertigo. Three weeks ago during a severe headache she suddenly became unconscious. This lasted for several minutes after which she was helped to bed. Since then she has had continuous headaches over the entire head. Patient is two months pregnant but thinks that that has nothing to do with her symptoms, judging from previous experiences.

Examination.—Right ear shows great destruction of membrana tympani. Tympanum dry. Left ear, two perforations in posterior half. Moderate amount of discharge having the odor of cholesteatoma.

Right.	Hearing Test.	Left.
L. C. V.	Voice	L. C. V.
	Weber	+
N.	Rinne	0
512	High	15 D.
	Low	—

Patient is very unsteady on her feet, sways from right to left and has some difficulty in maintaining the upright position. When walking turns off the to left unless she is careful.

Radical mastoid operation. Mastoid completely sclerosed. No antrum discovered. The position of the antrum could be

definitely made out but the cavity itself was filled with new bone. Tympanum filled with cholesteatoma. Promontory lacking in great portion, this causing a perforation in vestibule and first whorl of cochlea. This opening enlarged. Following operation vertigo was marked for ten days after which it gradually disappeared. Discharged well.

CASE 11.—Tiny B., school girl, age 14 years. Was brought to the University Hospital on account of a suppurative middle ear disease of the left side.

History.—Patient had scarlet fever at the age of 1 year. Both ears discharged at that time. At the same time a swelling appeared behind the ear. This was lanced and the wound soon healed. The discharge from the ear, however, persisted for about two years. From that time a little discharge appeared at times. About a year ago during a head cold pain in the left ear reappeared together with an increase in the amount of discharge. Two weeks previous to admission to the hospital, during a head cold pain reappeared and discharge ceased. Two days previous to admission a swelling was seen over the mastoid. About the same time child became much weaker. She has had at no times chills, nausea or vomiting.

Examination.—Shows a child fairly well nourished lying upon her right side, head markedly retracted, legs flexed on thighs, face flushed, eyes closed, mouth open, patellar reflexes absent, Kernig's sign marked. The heart while fairly strong when patient is lying down becomes irregular and weak during exertion. She answers questions intelligently but slowly and sometimes requires several efforts before she is able to finish her sentence. She protrudes the tongue fully but with some hesitation and unsteadiness. Temperature 102.8, pulse 140, respiration 24, leucocytes 17,000.

Local examination.—Right ear shows effect of a previous suppuration. Left ear, external auditory canal filled with foul cholesteatoma and pus. Lumen of canal narrowed by pressure from mastoid. Over the lower half of the mastoid is present a smooth swelling, very sensitive to pressure and showing slight fluctuation. This swelling extends beneath mastoid tip into the deep neck muscles.

A diagnosis of chronic suppurative middle ear disease, Bezold's mastoiditis with cholesteatoma and meningitis was made and a bad prognosis given. Careful questioning of the mother showed that the child had begun to complain of head-

ache only the day before and that all her bad symptoms had come on suddenly. Therefore it might be supposed that her meningitis had begun within the previous few hours, had come on suddenly, was progressing rapidly and might at that time be confined to an area that could be reached by operative measures. The writer could recall five cases of purulent otitis meningitis operated upon successfully in his own practice and therefore felt warranted in suggesting an operation as the only possible means to offer even the slightest chance of recovery. The child was therefore immediately prepared for operation.

Under ether anesthesia the usual mastoid incision was continued down over the area of swelling in the neck and several drams of foul pus and cholesteatoma were allowed to escape. Perforation through the inner surface of the mastoid tip had taken place on to the deep muscles of the neck. The mastoid cortex was completely removed and the mastoid cavity found of moderate size and filled with a true cholesteatomatous tumor having a well defined limiting membrane. This membrane had ruptured and allowed the escape of its fluid contents. Careful removal of the tumor showed the bony wall of the inner table destroyed by the disease so that the cerebellum was exposed over the area about the size of a silver dollar. Over this area, the membrane of the cholesteatoma was firmly adherent to the cerebellar tissue, i. e., a true subdural cholesteatoma. The dura in the neighborhood was part covered with foul purulent granulations. Those were removed and the brain uncovered until healthy dura was uncovered on all sides, except over a small area near the median line. The reason for not going further in this direction will be explained later. The lateral sinus was found uncovered for a distance of about an inch and a half but its lumen had not been invaded. The horizontal semicircular canal had been destroyed and the labyrinthine vestibule opened. A large amount of pus was secured from the posterior cerebral fossa. All pneumatic structure infected was then removed including the mastoid tip and the posterior root of the zygoma and the wall of the external canal, thus converting the middle ear, canal and mastoid into one cavity with smooth walls. The tympanic end of the Eustachian tube was then curetted in order to secure its future closure and a counter opening made in the neck for drainage. At this point, in spite of hypodermic stimulation and of

hot saline enemata repeated during operation the patient suddenly showed signs of complete collapse. Further operating was postponed and the patient returned to bed where she made a slow recovery from the anesthetic, regaining consciousness eight hours later.

9 p. m.—Patient was perfectly conscious but restless. She answered questions satisfactorily but slowly. Protrusion of the tongue showed paralysis of the left half of the tip. Opisthotonos became more marked with double optic papillitis.

May 10, 1907.—She suddenly became comatose and died.

Autopsy.—Brain showed slight tenseness of dura, considerable congestion of the pia, slight milky exudate over the left temporal lobe. Several drams of plastic exudate over left cerebellar area corresponding to field of operation and extending inward and downward so as to involve structures of the base. True cholesteatoma membrane adherent to pia, left lateral ventricle filled with flaky fluid. Choroid plexus congested. Cord shows several drams of cloudy fluid in the arachnoid of the lower dorsal and lumbar cord. The fact that the cholesteatoma occupied a position in immediate relationship with the internal auditory meatus, and that the changes in the vestibule were much greater than those in the semicircular canal system makes the inference possible that infection reached the labyrinth through the internal auditory meatus, and that the perforation through the horizontal semicircular canal took place from within outward, at least examination of the bone makes it evident that the changes within the vestibule antedate those in the canal.

TREATMENT.

In all cases in which labyrinthine suppuration has been suspected before operation or has been diagnosticated at the time of operation by the discovery of a sinus leading into the labyrinth the radical operation must be performed. Not only is this the only operative procedure by which the suppurative process can be dealt with but it is the only one that gives a satisfactory view of that part of the labyrinth wall through which infection has penetrated.

It has already been pointed out that infection reaches the labyrinth through the region of the semicircular canal, the oval window, the round window or the promontory. In order thoroughly to expose these areas, the last steps in the radical

operation must include the careful delineation of the three canals, the complete obliteration of the facial ridge even to the exposure of the facial nerve, the removal of the inner end of the anterior canal wall and of the external wall of the hypotympanum. The depths of the wound are then packed with gauze soaked in adrenalin in order that the field may be rendered bloodless and careful inspection of the labyrinth wall facilitated.

The complete exenteration of the labyrinth includes three distinct steps:

1. The entrance into the vestibule through the semicircular canal system.
2. The counter opening through the oval window.
3. The operation upon the cochlea through the promontory.

The operative field is so contracted, the relationship between the vital structures so intimate that a perfect orientation must be secured by work upon the cadaver before the exploration of the interior of the labyrinth can be attempted with safety to the patient or credit to the operator.

The entrance into the vestibule through the semicircular canal system is chosen as the first step in cases in which perforation through a canal can be demonstrated, as it offers a more comprehensive view of the vestibule than does the opening through the oval window. It is attended by less danger and less difficulty, it is less likely to affect the hearing disastrously and finally it sometimes reveals a localization of the pathologic process that renders further operative interference unnecessary. Removal of the semicircular canal system follows their satisfactory delineation. It is conveniently begun by shaving down with a curette or sharp chisel the superior margin of the horizontal semicircular canal in its posterior half.

This is continued until the canal is well opened. In the same way the posterior canal is widely opened. Then by means of the mallet and sharp narrow chisel the superior wall of the horizontal canal is followed directly inward until the vestibule is reached. The posterior and superior canals are then obliterated. The chief danger during this step in the operation lies in injury to the facial nerve. This is avoided by not interfering with the inferior wall of the horizontal canal which is in most intimate relationship with the Fallopian canal. I have demonstrated many times upon the cadaver that the chisel

must under no circumstances be allowed to impinge upon the wall of the Fallopian canal, nor to rest upon the crest of the previously opened horizontal canal while this canal is being obliterated, as the Fallopian canal will certainly be fractured. It is a wise precaution, I think, against injury to the facial to uncover the nerve for a considerable area of the horizontal portion in order that its exact position may be determined. Care is to be used in curetting the inner wall of the vestibule which is very thin, and rupture of which is attended by considerable danger, to both facial and auditory nerves. If painstaking examination seems to show that the disease is limited to the semicircular canal system further operative measures may be postponed. If, on the other hand, the interior of the vestibule suggests further extension of the disease or if a sinus leading through the oval window or promontory has been discovered the second step is undertaken. This procedure may be carried out as the first step in the operation if no perforation through canal is found. This consists in enlarging the oval window downward by means of a very sharp narrow chisel. In this way the posterior half of the inner tympanic wall is removed. With the greatest caution in order to avoid fracture of the inner wall of the vestibule into the internal auditory meatus, this opening is enlarged until satisfactory examination of the vestibule can be made.

The third step is the operation upon the cochlea. It is carried out in the cases only in which disease of the cochlea can be demonstrated. This, as J. D. Richards has recently pointed out, is the most dangerous step in the operation not only on the account of the most intimate relationship that the carotid artery, the jugular bulb, and the facial nerve bear to the field of operation, but especially on account of the position the very delicate and fragile modiolus that occupies the center of the cochlea. The modiolus is a hollow cone whose side forms the peripheral end of the internal auditory canal and whose apex is at a point an eighth of an inch in front of and on a level with the oval window. From his experiments and observations Richards concludes that the apex of the cone can be removed down to the first cochlear whorl without bringing the operative field and the intradural sac into gross communication and so rendering a fatal meningitis probable. Measurements of a number of temporal bone seems to prove to me that the internal auditory meatus terminates between one-

eighth and three-sixteenths of an inch internal to the promontory. Consequently to do more than uncap the pyramid must be exceedingly dangerous and is rarely necessary. The cochlea is opened safely by choosing a point one-fourth of an inch in front of the lower level of the oval window and chiseling downwards and forward very gently until a cochlear whorl appears. This is enlarged backward to the vestibule and forward as far as possible, chiseling as Richards advises always in the direction of the long axis of the pyramid. In this way two cochlear whorls are widely opened. This is followed by the cautious removal of the remainder of the promontory which permits inspection of the entire upper whorl, and almost all of the lower whorl. My experience upon the cadaver leads me to believe that in all cases in which indications are not absolutely plain, operative measures should cease at this point, although a small portion of the upper half lower whorl has not been seen. Even if caries at this point can be demonstrated it seems safer to allow this part of the cochlea to sequester rather than to remove it with the probability of opening the internal auditory meatus. Further removal of the upper portion of the cochlea also endangers the support of the facial nerve in the labyrinth. In cases in which this portion of the cochlea is perfectly loose it may be removed. My experience with operations upon the cochlea is limited to three cases, two of my own in which I successfully opened both whorls of the cochlea and removed the apex of the modiolus and one of Jansen's in which the cochlea was found to have sequestered and was completely and safely removed.

XVII.

CHONDROMA AND OSTEOMA IN THE FAUCIAL TONSILS.*

By Lorenzo Nelson Grosvenor, M. D.,

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Chicago.

It is my usual custom to make a pathologic examination of all the tonsils that I remove. In one pair I found something that played havoc with the microtome knife. On careful study of the sections I found considerable cartilage and bone growing within the structure of the tonsils.

A brief history of the case is as follows:

About the middle of last December, a nurse, 27 years of age, came in with her doctor and requested that I remove her tonsils. She gave a history of an attack of la grippe, associated with severe nausea, retching and vomiting, the month previous, but no history of trouble with the tonsils. In talking with her, I noticed a peculiarity of speech. She said that she had always talked so, but had paid no attention to it. On examination, the right tonsil was found larger than the left, although neither was especially large. In looking for the cause of her speech defect I could find nothing abnormal with the postnasal space or larynx, but the soft palate proved to be very firm and unyielding when she swallowed or pronounced certain letters. The only part of the soft palate that appeared to move was a small triangular area, about 4 mm. across, just above the uvula. The nurse and her doctor thought that there would be some relief of the difficulty of speech, if the tonsils were removed.

Under local anesthesia, swabbing with 10 per cent cocain, and hypodermic of 2 per cent cocain in 1 to 2000 suprarenalin, I performed a tonsillectomy. First freeing the right tonsil from the pillars and the supratonsillar membranes, with the

*Presented before the Chicago Laryngological and Otological Society, April 14, 1908.

Pierce pillar knife, then applying the newly modified Boettcher-Farlow snare, the left tonsil was enucleated with the Pierce tonsil knife. Both tonsils were removed without any difficulty, but with the usual amount of hemorrhage. She stood the operation well, but before morning her former gastric troubles started up, with severe nausea and retching, which persisted for several days.

In gross appearance, these tonsils were not like the usual tonsil, large, compact and plump, but rather small with long thin processes, separated by broad deep crypts. The right tonsil, under the microscope, presents a broad band of cartilage and bone in the capsule, which extends into the broad fibrous septa that run down into the processes of lymphoid tissue, on each side of a deep crypt.

In studying the sections, of many different levels, the cartilage and bone were found to curve in, crowding through the lymphoid tissue, to meet each other, thus forming an irregular circle about 4 mm. in diameter, inclosing much lymphoid tissue surrounding the base of a crypt. There was always a dense fibrous band separating the cartilage or bone from the lymphoid tissue. There were other abnormal conditions about this tonsil. On the lateral surface, beyond the cartilage, there was considerable muscle structure. On the lower end of the tonsil there was a secretory gland, probably salivary. Several lobes of this gland crowded up, back of the lymphoid tissue, external to the lowest crypt, and crowded in between the cartilage and lymphoid follicles, that extended down into one of the processes. At the upper end of the tonsil a band of pure cartilage branched out from the cartilaginous circle and extended up along the capsule.

The left tonsil was sectioned in such a way as to make cross sections of several crypts. Back of these was a large triangle, 4 mm. across, made up of a broad fibrous band, the base of which forms the capsule, the point crowded up between masses of lymphoid tissue. Inclosed within this triangle were two smaller masses of lymphoid tissue, separated by a band of connective tissue. In this broad fibrous triangle were bands of cartilage, in which were seen many areas that had gone on to ossification.

On careful study of the cartilage and bone in these tonsils, I found the cartilaginous areas presented the typical structure of hyaline cartilage with lacunae, each inclosing a cartilaginous

cell. Closely surrounding the cartilage was a band of fibrous connective tissue forming a perichondrium, and, with certain stains, notably Verhoeff's or Mallory's, one can see the surrounding fibers entering into and becoming a part of the cartilaginous structure. In many areas of the cartilage there were centers of ossification, which had developed into true bone, with their Howslip's lacunae lined with osteoblasts. With proper staining and careful study, one can see the apparent transformation of fibrous into cartilaginous tissue, passing on into bony structure. In some areas the bone is entirely surrounded by fibrous tissue, forming a periosteum, while in other areas the bone is in the midst of cartilage. The new elastic tissue staining method, just published by Verhoeff, demonstrates the formation of the cartilage and the development of ossification better than any other stain. Mallory's reticulum stain demonstrates the bone structure best.

A number of writers, notably Newcomb, Roe, Richards and Stirling, have reported difficulty in removing tonsils with the tonsillotome, finding it necessary to resort to bone cutting forceps to complete the removal of the tonsils. On examination, a single nodule of bone, supposedly from the styloid process, was found protruding into the tonsil. To reach the tonsillar region, the styloid process, which extends usually downward, forward and inward from the under surface of the temporal bone to an extent of two to three centimeters, would have to be elongated to more than five centimeters and curved forward and inward. Microscopically, this would present a single nodule of cartilage or bone and could be traced out through the structure of the tonsil. Palpation of the lateral wall of the pharynx would reveal the remaining stump of the styloid process, between the angle of the inferior maxilla and the vertebral column.

Another group of writers, namely, Carter, Kanthack, Lubarsch, Petroff, Pollak, Reitman and Toepfer, believe that the formation of cartilage and bone in the tonsils is due to metaplastic changes, of fibrous tissue into cartilage and then into bone. In the study of the tonsils in my case, the appearance suggests the possibility of just such a change, but to confirm this theory, there should be a definite clinical history and pathologic findings of chronic inflammation, such as these writers report, but which was lacking in my case. Senn, in his book on "Tumors" (1900) says: "The metaplastic theory con-

cerning origin of bone is no longer tenable." "Tissue begets tissue of its own kind." There must be a matrix of embryonal cells as origin of development.

In the many cases reported by Coakley, Diechert, Halkin, Nosske, Orth, Roth, Ruckert, Walsham and Wingrave, the consensus of opinion is, that the cartilage and bone found in the tonsils, originated in abnormal development of cartilaginous rests of the second branchial arch. In the embryologic study of this second branchial arch, there is the dorsoventral cartilaginous bar, called "Reichert's Cartilage" made up of five segments. The stapes, by some, is supposed to be developed from the first segment. The second segment forms the base of the styloid process. The third segment forms the lower part of the styloid. It undergoes ossification later than the second segment and does not acquire osseous union with it, until adult life. Sometimes it remains separate throughout life. The fourth segment remains fibrous and forms the stylohyoid ligament. The fifth segment becomes the lesser cornu of the hyoid bone. The tonsil develops as masses of lymphoid tissue about an evagination of the lateral wall of the pharynx, in the third month of fetal life.

Cohnheim referred every tumor to its proper embryonic layer and claimed that a tumor never had its origin from mature tissue, but always developed from a matrix of embryonic tissue. He believed that during the process of cell differentiation in the embryo, groups of cells not utilized, or displaced, in the growth of the embryo, were arrested in their further development and remained latent until aroused to activity, when their proliferation resulted in the formation of a tumor. Hence, the matrix of embryonic cells is the essential cause of tumor formation, without which, all intrinsic and extrinsic causes are inadequate to produce a true tumor.

In these cases, then, where cartilage and bone have been found in the tonsils there must have been a displaced or unused matrix of embryonic cells. The second branchial arch furnishes us with the possible source of embryonic cells of this nature. In the same manner, we find cartilage in the parotid glands and about the external ear that originated from embryonic cells derived from the first branchial arch.

In the study of my case, I believe the following conclusions are justifiable:

1. The cartilage and bone are not part of the styloid process,

for we cannot palpate the stump of the styloid between the inferior maxilla and the vertebral column. Here, too, there is not a single nodule of cartilage or bone, but a ring or triangle.

2. The condition is not one of metaplastic changes, for there was no clinical history or pathologic findings that would cause such changes.

3. The logical conclusion, therefore, must be that the cartilage and bone found in these tonsils, was developed from a matrix of unused or displaced embryonic cells derived from the second branchial arch.

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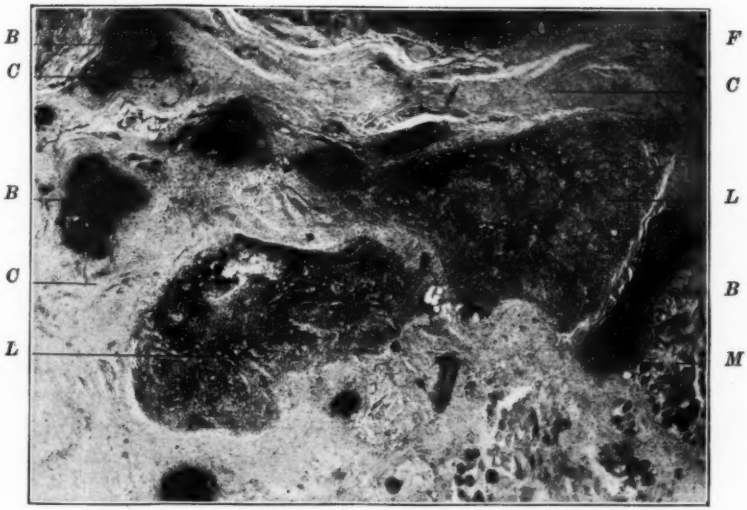

FIG. 1.—Fibrous triangle with areas of cartilage and bone. B—Bone; C—Cartilage; F—Fibrous connective tissue; L—Lymphoid tissue; M—Muscle. Hemotoxylin-eosin stain. Magnification 25.

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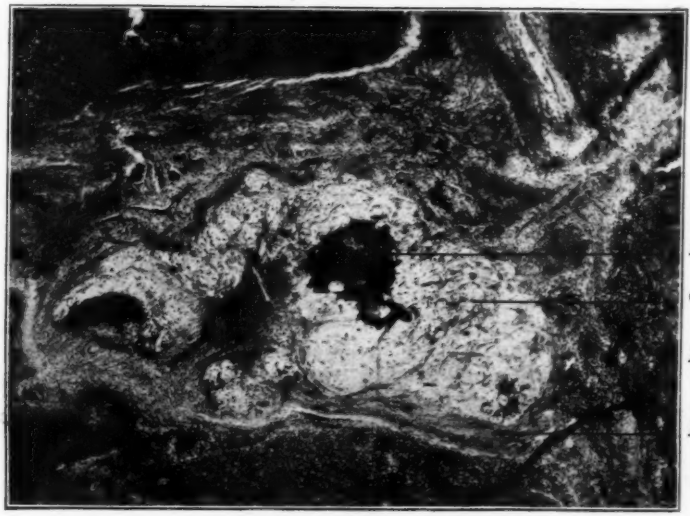

FIG. 2—Mass of cartilage and bone. B—Center of ossification. C—Cartilage. L—Lymphoid tissue. Verhoeff's Stain. Magnification 55.

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XVIII.

DEVELOPMENTAL ABSENCE OF THE OUTER RIGHT SPHENOIDAL WALL OCCUPIED BY A VEIN COMMUNICATING DIRECTLY WITH THE CAVERNOUS SINUS. OPERATIVE FATALITY AND AUTOPSY.*

By Francis P. Emerson, M. D.,

Boston.

In the surgery of the accessory sinuses, brilliant accomplishment has been attended by unavoidable mishap. To overcome this or perfect the operation, various procedures have come to be known by their originators until today half a dozen definite and different plans are offered to accomplish the same end, some of them conservative and some the most thorough and radical that modern surgery can offer. The more that one studies anatomic specimens in connection with the sinuses, the more he is impressed with the large number of abnormalities and the necessity of exact measurements and familiarity with the various cavities of the head. Vital centers that are separated from pus cavities only by cribriform osseous plates lined by degenerate tissue are not ideal fields for operation. After several years of work in draining the sphenoid without anything suggesting an accident, the following condition was found and its attempted correction attended by a fatality:

In June, 1904, Miss L., 21 years of age, born in Massachusetts, and a school teacher by occupation, was referred to me by Dr. B. for treatment, with the following history:

Past History.—Severe attack of diphtheria at 5 years of age. Light attack of measles. Catarrh since childhood. The act of swallowing food was accompanied by gagging and vomiting which released a thick tenacious mass from the posterior nares. When actual vomiting did not occur, pharyngeal irritation excited hawking so that she has to leave the table. To

* Presented before the annual meeting of the Eastern Section of the American Laryngological, Rhinological and Otological Society, Boston, January, 1908.

avoid this she has been in the habit of using an anterior douche previous to each meal. There is pain over the bridge of the nose.

Examination.—Anterior nares normal, except an enlargement of the left anterior turbinal cell of the middle ethmoid. Posteriorly there is a tenacious mass covering the entire pharyngeal vault. The posterior wall of the pharynx is glazed and dry. A small amount of dry secretion covers the anterior end of each middle turbinate. A bullet pointed probe shows a carious area in both sphenoids. The antra and frontal sinuses illuminate normally. General condition good.

July 5th.—The right anterior sphenoidal wall was removed with the Grünwald forceps, and the anterior end of the right middle turbinate with scissors and snare.

April 15th.—The left anterior sphenoidal wall was removed, together with the anterior end of the left middle turbinate. A carious area on the floor and inner wall was curetted.

March 24th, 1905.—No symptoms except dryness of the pharynx.

November 17th, 1906.—Came back complaining of a return of former symptoms, but less marked. A large cell was found containing pus, in the remains of the left middle turbinate which was opened and drained with biting forceps.

March 25th, 1907.—A thin tenacious grayish coating was found still present over the pharyngeal vault. Patient said that this still troubled her about twice a week. A thorough examination was again made and in the right sphenoid a carious area was felt on the outer wall with a soft mass posteriorly. On account of the previous operation this was taken to be polypoid, and but little force was used in probing, for fear of exciting hemorrhage. The middle turbinate and the anterior wall of the sphenoid had already been removed. A curette was introduced behind this presenting mass and gently withdrawn, but was followed by a profuse venous hemorrhage and a convulsion. The patient immediately became unconscious. A rapid tampon, after the patient had fallen in collapse on the floor, controlled the hemorrhage externally, but symptoms of brain pressure supervened, and she was removed to the Charlesgate Hospital, where she died at 11 o'clock, seven hours after the accident.

Autopsy by Dr. Leary, of Tufts Medical School, in the presence of Drs. Arnold, Courtney, Swan and others. A hemor-

rhagic clot filled the calvarium. A large varicose vein was found to occupy a dehiscence in the outer wall of the right sphenoid, which communicated directly with the cavernous sinus, and was the cause of death. The remaining ethmoid cells were everywhere necrotic, and about one-half ounce of mucopurulent secretion was found.

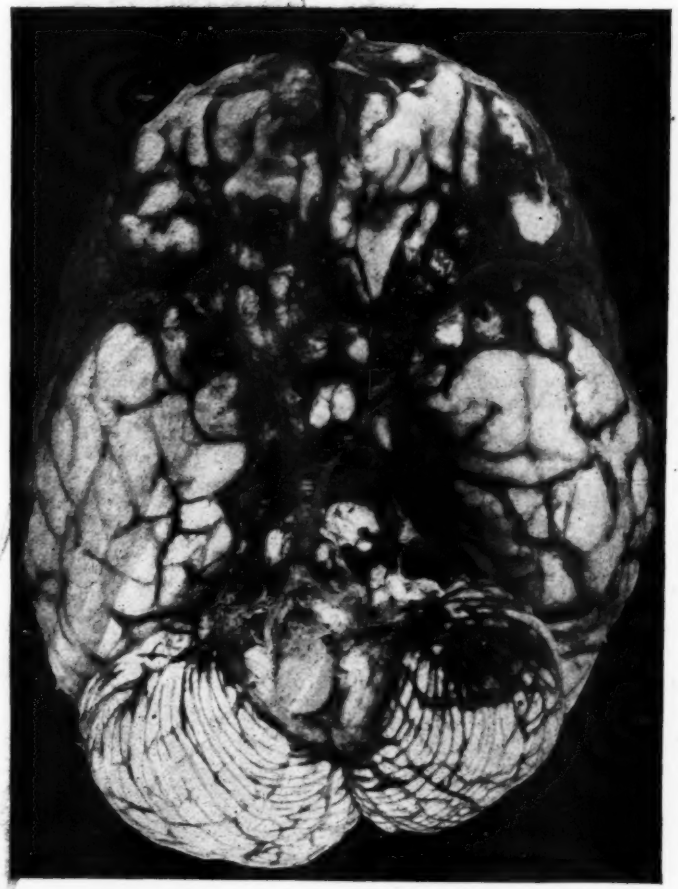
It was my custom in my early work on the sphenoids to break down the anterior wall (i. e., enlarge the normal opening with a Blake mastoid curette) until I found that this was apt to be followed by a rapid closure anteriorly before the cavity was dry. Latterly the Grünwald forceps that bite from behind forward, have proved more satisfactory and the drainage alone cures the case clinically. This is fortunate for if one studies a large number of sphenoid sinuses on the cadaver, he is at once impressed with the extreme thinness of the superior and outer walls which indeed may be cribriform, or so thin as to be readily seen through. The outer wall in addition may project into the general cavity so that it might be readily broken down in the radical Killian operation, unless one were working by careful measurements. It is easy to conceive that the dragging forward of a polypoid mass, even though there were no break in the osseous structure, might be accompanied by disastrous results.

While clinical experience has taught us that the sinuses are rarely involved separately, there are a large number of cases that cease to give symptoms if properly drained intranasally. It is usual to associate disease of the sphenoid sinus with a glazed pharynx, a thick tenacious mass in the vault and dried secretion on the anterior surface of the middle turbinate. In women this is usually accompanied by an occipital or vertex headache. The diagnosis is readily confirmed by a small bullet pointed probe passed directly back to the pharynx through a line that passes midway between the anterior end of the middle turbinate and the septum. If it does not at once enter the osteum, slight elevation or depression usually at once locates it. The anterior end of the middle turbinate need not be disturbed unless diseased. It is unnecessary for diagnosis and generally is not required for treatment. The small probe with the shoulder which its bullet point gives it, will enter any osteum without special curve or measurement, as it can be manipulated very delicately, the bare bone being at once felt. Emphasis should be laid on the fact that removal of the an-

terior wall is all that is necessary to relieve symptoms. Cleansing is not only unnecessary, but is usually followed by a distressing headache. Other measures are dangerous and not needed. The removal of the anterior wall should be accomplished by the instrument producing the least trauma, and is therefore followed by the least reaction and tendency to fill in before the cavity is thoroughly drained.

In the case cited, it was my intention to insert the curette behind the mass, entering from the inner side and draw forward. In doing so I did not feel any sensation of bony contact, which one is apt to recognize however slight the contact of a steel instrument with an osseous surface.

Dr. Leary's careful examination of the recent specimen showed an absence of the outer wall, and a large vein which communicated directly with the cavernous sinus, and which was mistaken for polypoid tissue. Later, after the specimen had been prepared by the prosectors, an undoubted fracture was found in the outer wall, and whether this was made at the time of the operation, or later in its final preparation, cannot be determined.



Subpial hemorrhage from vein in the outer wall of the sphenoid communicating with the cavernous sinus.

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a. Developmental abscess of the outer wall of the sphenoid. Fatal hemorrhage from a large vein communicating with the cavernous sinus.

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XIX.

REPORT OF A CASE IN WHICH DEFICIENT CEREBRATION WAS A PROMINENT SYMPTOM IN OTITIS MEDIA PURULENTA CHRONICA—CURED BY A RADICAL MASTOID OPERATION.

By D. W. Layman, M. D.,

Indianapolis.

A special form of diminished power of mental application, termed by Prof. Guye "aprosexia," is frequently noticed in children affected with postnasal growths, with or without exudate catarrh of the middle ear. We understand the cause of cerebral deficiency in these adenoid children, and know that under appropriate operative treatment such cases become normal, or are at least greatly improved, but I have never met with a condition such as aprosexia existing purely as the result of an otitis media purulenta chronica. It is familiar knowledge among otologists that such symptoms as "a marked change in the mental state, consisting of dullness and hebetude, seeming lack of thought or great slowness of thought, desire to be left alone and to keep the mind at rest, imperfect power of attention, defective memory of recent events," are all usual symptoms of intracranial lesions when they appear in cases of chronic purulent otitis media.

I wish to show, however, that the above symptoms can occur in a case of chronic purulent otitis media not intracranially complicated, but complicated with a chronic mastoiditis. I, therefore, present the history and treatment of the following case occurring in my hospital practice:

Harry H., aged 7 years, had scarlet fever when a baby, or a very young child. This was complicated with an otitis media purulenta. So far as could be learned, he had suffered from suppuration in the left ear since the attack of fever. When I first saw him the aural discharge was profuse and of very fetid and caseous character.

An aural examination in the fall of 1905 showed destruction of all but the upper part of the tympanic membrane; the handle

of the malleus projecting into the perforation. On the posterior half of the tympanic cavity there was a polyp attached with a narrow base. Examination of the throat and nasopharynx revealed diseased adherent and submerged tonsils and small adenoids. Hearing was impaired, but not to a marked extent.

The ordinary symptoms of aprosexia, difficulty in sustaining attention, dulness and slowness in school, were noticeable, but attributed at that time to the presence of the postnasal growth and submerged tonsils. Subsequent removal of these growths, however, proved that this was not the case. The growths were removed in the spring of 1906. The following year the child was quarrelsome, ugly and hard to manage in kindergarten; in fact, the teachers told me they dreaded the care of the boy on account of his ugly disposition. Besides, he was slow to learn, and evinced a seeming lack of thought and slowness of intellect, and showed a desire to be left alone.

His ear was treated with antiseptic irrigation and instillation of drops after the removal of the polyp. As this treatment proved to be of very little benefit, I decided to perform a radical mastoid operation. This was done in June, 1907. The operation was followed by an uneventful recovery.

A word or two about the operation.

The Schwartze-Stacke operation was performed, that is, the antrum, tympanum, attic and external auditory canal were converted into one cavity by removal of much necrosed bone, granulations, cholesteatoma and the remnants of the membrana tympani and ossicles, except the stapes. The natural meatus was enlarged by making an upper and lower flap of the cartilaginous meatus, by carrying a horizontal incision outward into the concha a distance sufficient to insure a good opening. The upper flap was sutured back over the dome of the excavation at superior rim of bone cavity. The lower flap turned down and sutured to the anterior lip of the curvilinear wound below. The mastoid wound was closed behind and healed by primary union. The wound was subsequently dressed through the enlarged meatus, and the entire surface dermatized nicely.

The operation was not performed with any idea of improving the child's disposition or mental state, but only with the view of ridding the patient of the suppuration in the temporal bone. The nurse at the hospital called my attention to the

improvement in his mental state just prior to his leaving the hospital for the Orphans' Home. Afterwards I interested myself in this phase of the case, and asked the matron to notice whether she detected any change in the conduct of the boy. Three months after the operation she told me that Harry was like a different child, that he was bright, cheerful and took an interest in everything, and she said that the teachers reported that he was easy to manage and showed none of his old ugly disposition, and that he learned in school as readily as the average scholar. There was no improvement in hearing after the operation, but there apparently seemed to be, as he was very attentive.

We know that enduring forms of headache are caused by old chronic suppuration of the ear, especially with collection of exfoliated epithelium and granulation tissue in the cavities of the middle ear and spaces inside the temporal bone. Politzer mentions a feeling of pressure and heaviness in the head, or obstinate headache, as symptoms of chronic middle ear suppuration, especially in those cases complicated with mastoid disease. It is presumed that the symptoms mentioned by Politzer especially refer to cases of chronic purulent otitis media in adults. Children would less readily complain of a feeling of pressure and heaviness in the head, or obstinate headache; then, why could not these similar lesions in children produce symptoms which manifest themselves by all the signs of deficient power of mental application as in the case reported.

It might be asked, are these symptoms of deficient cerebration the result of a feeling of fullness in the head, or irritation due to more or less pressure on the brain by close proximity of the suppurating cavities, upsetting directly or indirectly the mental balance or nervous state, or, are they due to absorption of the toxines of pyogenic bacteria?

XX.

THE TREATMENT OF INTUMESCENT RHINITIS BY A SUBMUCOUS METHOD.

By Henry Horn, M. D.,

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(From the Universitatspoliklinik fur Ohren-, Hals-, und Nasenkranke, in Bonn a-R. Director: Geh. Med.—Rat. Prof. Dr. Walb.)

Intumescent rhinitis is of itself not a pathologic entity but the term clearly conveys to us the picture of a clinical condition which is of very common occurrence indeed. The etiology also is not clear, many throwing the causal burden entirely on the shoulders of a general bodily disturbance, such as alcoholism, circulatory, heart and kidney diseases, scrofulosis, anemia, etc., while others consider the condition purely a local one.

Probably the middle ground is nearer to the real truth of the case, and it goes without saying that where a physical examination revealed some general complaint, one would endeavor, as far as possible, to correct this condition in connection with the local treatment. On the other hand very few surgeons are in sympathy with that school which hold local measures, especially the employment of the galvanocaustic, unnecessary, and confine themselves to a general constitutional treatment only.

The brilliant results which have been achieved by the use of the galvanocaustic in the amelioration of this complaint are not to be denied. Where failures are found, one can usually explain them by a lack of a proper selection of the cases.

That every case of intumescent rhinitis can be benefited by the use of the galvanocaustic, and that every case of hypertrophic rhinitis cannot so be helped, places us in the position of asking what the dividing line is between these two conditions. Pathologically there is none, clinically an easy rule can be stated. If a turbinate with a smooth mucous membrane surface is easily reduced in size by cocain, it is favorable for

the procedure. If it is slightly or not at all reduced, it is usually a case for a cutting operation.

The term smooth mucous membrane surface is used simply to exclude all polypoid degenerations of the membrane, all hypertrophies falling under the classification of raspberry (himmbeerartig), grape like (traubenartig), plicated (lappigenhypertrophy), which of course do not fall within the province of this paper, and are only to be treated by cutting or snaring operations.

In the splendid work of Ingals and Friedberg, on the "Treatment of Intumescent and Hypertrophic Rhinitis" in these ANNALS for March, 1906, we secure such a clear picture of the etiology, general course and clinical symptoms, that it is quite unnecessary to cover the same ground again. In a large clinic, where the general hygienic conditions of the patient are exceedingly unfavorable, where the mucous membrane tone is notably below par, where the material is enormous and constantly changing, where one is never certain of the patient returning for after-treatment; the disadvantages of the usual methods of cauterizing the inferior turbinate become quickly apparent.

The complications which depend far more upon the after-treatment and hygienic surroundings of the patient, than upon the operation itself, have been far more common with us than is shown by the statistics of the above mentioned authors, who perhaps included in their results many cases handled in private practice.

Not a month passes but one or more patients present themselves in the clinic with a septum-turbinate adhesion. The history almost invariably given is: that the nose was "burnt" because it was "stopped up," and that they had "never returned for after-treatment." The adhesions occur almost invariably in males and especially in persons with narrow noses and septal deviations. This is notably the case in all statistics, and seems to depend on the occupation, which by the inhalation of dust, chemical vapors, etc., brings about a local irritation, and rather speaks against a constitutional disturbance as the sole cause. Narrow noses with septal spurs, ridges or deviations are especially prone to lead to poor results by the old methods of cauterization.

Angina lacunaris, as first mentioned by B. Fränkel, is an exceedingly common complication, coming on about the third

day and often occurring in spite of the most careful after-treatment.

Septum-turbinate adhesions are among the most disagreeable of all the complications which have their origin in this procedure, and is by no means as infrequent as the statistics of the above mentioned authors would indicate. In narrow noses combined with sharp crests, an accidental burning is sometimes unavoidable and the subsequent results far from satisfactory. Just here another point arises which has only made itself felt since the introduction of the submucous resection of the septum; no septum operation is pleasant where the mucous membrane is scarred through a previous operation or an accidental burning of its mucous membrane, and no septum operation fulfills its function if we leave the swollen turbinates untreated. The preliminary cauterization of the turbinates seems the rational procedure.

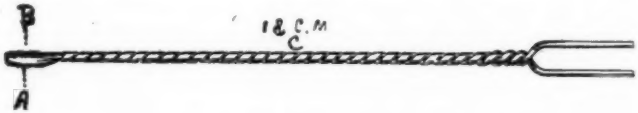
The submucous caustic of the inferior turbinate, when properly carried out offers an ideal solution out of all of these difficulties.

Some years ago a procedure, generally ascribed to Grünwald, was proposed along these lines. This consisted in entering the anterior end of the turbinate with a glowing point and gradually undermining the mucous membrane as far back as possible. The practical result was that at the point of entrance a large burn was made, and at the posterior end a second perforation resulted. If the membrane was at all adherent at any point, a third perforation was made, until finally during the healing process, the intervening mucous membrane would slough away and an open wound result.

The next step was reported from the Hajek Klinik in a short note of ten lines. Here the anterior end of the turbinate was entered with a knife, the membrane undermined, and an ordinary pointed burner introduced cold and withdrawn hot. This procedure also had its disadvantages. The sharp knife often perforated the membrane in the middle third and at best only cut through the intumescent vessels, leaving an unsatisfactory passage for the point to follow. If the swelling was not marked or if, as it often happens, the middle third was slightly adherent, the unprotected membrane would be burned through and again we would have to deal with an open wound.

The submucous method, about to be described, does away with all these disagreeable features and has as well the following great advantages:

1. Complete healing takes place in from three to five days.
2. The swelling after the operation is practically nil.
3. The mucous membrane remains intact throughout its entire length.
4. The wound is aseptic. The entrance point closes of itself.
5. Adhesions with the septum are impossible.
6. The physiologic functions of the mucous membrane are preserved.
7. The burning takes place only in that tissue which it is intended to destroy: The cavernous tissues of the turbinate.
8. The shrinkage is complete along the entire length of the turbinate, and an ideal and permanent air space is the result.
9. So far no complicating tonsillitis, middle ear inflammations, or adhesions have been observed.
10. Burns of the septal membrane are absolutely avoided.
11. The disagreeable smell of the burning tissues and the occluding smoke are done away with.
12. If the patient fails to return for treatment no harm can result.



Operation.—A cotton tampon moistened with a 20 per cent solution of cocain is laid in the nose for a few minutes until the turbinate is entirely free from feeling as tested by the knife point. If the intumescence is not great and the cocain promises too great a shrinking, a similar solution of alypin can be used.

A small perpendicular incision is made through the membrane at the anterior end of the turbinate. With a Freer septum elevator, the mucous membrane is dissected free from the bone as broadly and as far back as possible. The use of a large elevator or a knife is not to be recommended, as a perforation is easily made.

The form of the burner is of great importance. As shown in the illustration, I use the so-called aseptic burner (C), 12 cm. long, with a protected burning wire. The part (a) is of very heavy platinoiridium wire 0.7 mm. in diameter and 1 cm. long. The part (b) remains cold during use and protects the overlying mucous membrane. The rheostat is set beforehand at

almost a white heat, as the blood under the upraised membrane causes a rapid cooling of the point, thereby allowing it to become fastened to the membrane and perhaps causing a tear.

The burner with the platinum turned toward the bone is introduced cold to the posterior end of the pocket formed by the upraised membrane, withdrawn a few millimeters, to prevent perforation of the end pocket, and the current turned on. The burner, always closely and firmly pressing the bone, is slowly withdrawn. Just before the entrance is reached the current is shut off, and the burner quickly removed.

The overlying mucous membrane, being protected by the form of the burner, is not injured, but immediately and firmly contracts and subsequently becomes firmly attached to the bone. The vessels are permanently destroyed thus greatly protecting against a recurrence. Over a year has elapsed since some of my first cases were operated by this method and a present examination shows the same condition as was seen two weeks after the operation.

ABSTRACTS FROM CURRENT OTOLOGIC, RHINOLOGIC AND LARYNGOLOGIC LITERATURE.

I.—EAR.

Report of Cases of Brain Abscess.

F. E. KITTREDGE, Nashua, N. H. Case one was a full-blooded Indian, 9 years of age, in which the brain abscess followed about a month after an operation for a purulent mastoid and an epidural abscess, recovery from which had occurred. This was followed by severe headaches, violent attacks of vomiting, slightly subnormal temperature and a pulse of about 60. The original incision being reopened, the dura was found to be in a healthy condition but somewhat bulging. Laying open the membrane, went in about one inch and a half with a needle and pus was found one and one-half inches deep in two pockets. Both cavities were drained with gauze; about one and a half ounces of pus being evacuated. The case did well for three weeks when meningeal symptoms developed, followed by unconsciousness and death. After the development of the meningeal symptoms, a thorough examination was made with the finger which seemed to break into the wall of the lateral ventricle and upon the withdrawal of the finger, water and pus of considerable quantity, both came away. The author estimates that one-third of the hemisphere had disappeared.

CASE 2.—Male, 30 years of age. Simple mastoid first done. Afterwards had a severe convulsion followed by drowziness, vomiting, temperature of 99, pulse 60. Brain abscess diagnosed, trephined about one inch above the zygomatic ridge; bulging dura, pocket of pus one-half inch from the surface. Bone opening enlarged and good drainage secured with gauze. Recovery about three weeks after operation.

CASE 3.—Male, 27 years of age, slight discharge but no mastoid symptoms. Temperature 102 to 105, delirium, unconsciousness, meningitis; brain abscess suspected. Trephined through the right temporo-sphenoidal region just above the ear, dura opened, brain tissue incised, two ounces of pus evacuated. Death in three days. No relief having occurred except of the headache.

CASE 4.—Male, 16 years of age. Severe headache, tempera-

ture 101, pulse 55; mastoid symptoms. Operated for the mastoid. Headache no better, slow pulse, vomiting. Brain abscess diagnosed. Dura opened after trephining; no pus found in the zygomatic region. Trephined over the cerebellar region and explored with trocar, nothing found. Symptoms not improved at all and after several days with a temperature ranging from 98 to 100 and pulse 45 to 55, further search for pus was made, by opening the original mastoid cavity between the semicircular canal and the anterior part of the cerebellum; no pus was found. The openings were packed with gauze and there was considerable discharge of serum. Headache improved, pulse went to 60. A day or two later there was marked nystagmus in both eyes followed by temporary paralysis in the left side of the face. Recovery took place. Whether this last particularly belongs with the brain abscess cases may be a question. *Richards.*

Pathologic Anatomy of Labyrinth Inflammation Extending From the Middle Ear.

W. LANGE (*Beiträge z. Anat., Phys., Path. u. Therapie d. Ohres. der Nase und des Kehlkopfes,* Bd. 1, Nos. 1-2) reports in considerable detail the histologic findings in 14 cases of labyrinth suppuration. He found that in 9 of these cases the point of primary infection was through the semicircular canals; in 4 through the oval and round window; and in 1 through both the semicircular canal and the windows. In acute cases the windows seemed more frequently to be the primary points of entrance than the semicircular canal. Polyps and excessive granulations are most frequently the predisposing conditions. Cholesteatoma by causing erosion of the semicircular canal open a gateway through which an acute infection may at any time find entrance to the labyrinth. As to extension of the disease within the labyrinth, Lange found that in all his cases where the membranous labyrinth was necrotic, the ductus endolymphaticus showed more or less involvement. The soft parts of the ductus were generally necrotic almost to the external aperture of the aquaeductus vestibuli. In 4 cases the propagation of the inflammation through the cochlea was stopped by old inflammatory deposits in the scala tympani, and in one case recent changes had completely obstructed the lumen of the scala tympani. In 8 of the 14

cases pus was found in the internal auditory meatus The function of the facial nerve was destroyed in only one case. There are a good many references mentioned in this article but they are all from German literature. *Wood.*

Closure of Bone Defects After Antrum Operations.

A. Passow (*Beiträge z. Anat., Phys., Path. u. Therapie d. Ohres. der Nase und des Kehlkopfes,* Bd. 1. Nos. 1-2). For the closure of old bone fistula following mastoid operations, Passow suggests the following technique: The scar is split throughout its length by an incision behind the ear, following the first operation. This incision should be carried completely through the depressed cicatrix, and the skin elevated from the wound. The bone wound is then carefully freshened with a curette. The skin around the wound is undermined for a considerable distance and carefully separated from the connective tissue and periosteum. The periosteal flaps are now carefully raised, one in front, one behind, the wound. The flaps whose bases are below must correspond in size to the breadth and depth of the wound. These flaps are now sutured in the middle line with catgut, and pushed into the cavity in the bone. The layer of connective tissue and periosteum posterior to the wound is thin, so that in this direction the broadest possible flap should be obtained. The flap from near the insertion of the ear is very narrow but generally rather thick. The scar tissue of the skin-wound is removed with scissors and the skin edges then brought together, preferably with Michel's hooks. This method of utilizing the periosteum, according to Passow, rarely fails to close in these post-operative defects in the bone. *Wood.*

Anatomy of Congenital Deafness.

Ferdinand Alt (*Monatsschrift für Ohrenheilkunde sowie für Kehlkopf. Nasen. Rachenkrankeiten,* February, 1908) reports a case of congenital deafness, in which he made a careful and complete histologic examination of the ears. The patient had died from tuberculosis. The case was one of complete deafness on both sides. The middle ear was practically normal, and the labyrinth followed out in general the ordinary scheme. The terminal elements of the nerves were only par-

tially developed, and further had subsequently undergone a marked atrophy. This condition was seen in the vestibules in all of the sensory epithelium of the cochlea, and also there was a collapse of the membranous labyrinth. The nerve itself in the region of the internal auditory canal showed less atrophy than in the individual nerve canals and the sensory epithelium. There was no sign of any inflammation either recent or old.

Wood.

Retroauricular Opening After Radical Mastoid Operation.

ERNST WINCKLER (*Arch. f. Ohrenheilk.*, Bd. 75, Nos. 1 and 2) makes a plea for the establishment of a permanent opening back of the ear, following the radical operation. He says it is especially indicated if there is any labyrinth complication, fistula of the semicircular canals, or disease of the promontory, making a careful observation of these structures necessary after the operation. Further, it is very useful when the cells surrounding the Eustachian tube are involved, and also in cases of complete exenteration of the middle ear which before they have healed come into the hands of another physician. This retroauricular opening affords the second physician a good chance to look over the operative wound. In tuberculosis of the middle ear the retroauricular opening is absolutely indicated, and also where there is cholesteatoma present.

Winckler gives in detail his method of operating for the establishment of a permanent opening. *Wood.*

Acute Labyrinthitis.

G. ALEXANDER (*Archiv. für Ohrenheilkunde*, Bd. 75, Nos. 1 and 2) reports four cases of inflammation of the labyrinth following the radical mastoid operation. The chief symptoms were nystagmus toward the diseased side, dizziness, and a loss of equilibrium coming on within 24 hours after the operation. Also, in two cases there was some meningitis. The dizziness and loss of equilibrium cleared up in a few day, but the nystagmus generally persisted for a week or more. The rapidity of the cessation of the symptoms differentiated these cases from purulent labyrinthitis, and it is most probable that the condition present was a circumscribed serous labyrinthitis. While in none of the cases was the labyrinth visibly injured

before or during the operation, it is most probable that the unavoidable trauma of the middle ear was the important etiologic factor. *Wood.*

Bier's Hyperemia Treatment in Acute Middle Ear Suppuration.

F. ISEMER (*Arch. für Ohrenheilkunde,* Bd. 75, Nos. 1 and 2) reports in detail nine more cases of otitis media treated by Bier's passive hyperemia. From a study of these cases he arrives at slightly different conclusions than formerly. He says that treatment of otitis media with passive hyperemia is not void of danger, as in employing this therapy the symptoms became so markedly lessened that it is not an easy matter to know when to institute surgical interference. Hence he does not advise its use by the general practitioner. Streptococcic and diplococcic infections are especially dangerous. The hyperemia works better in staphylococcic infections and especially in cases with mastoiditis where there exists a cortical fistula. Here an incision through the soft parts gives a chance for the pus to escape. Relief from pain is the most striking result of the hyperemia treatment, but this in some cases may be exceedingly dangerous. This treatment should never be used in cases with intracranial complications.

Wood.

II.—NOSE.

Pansinusitis Nasalis with Fatal Course.

H. J. L. STRUYCKEN (*Tydschr. v. Geneesk.,* May 11, 1907) reports the following: Consulted in October, 1900, by a 48-year-old man for right-sided otorrhea, headache, nasal obstruction and mucopurulent discharge from nose and postnasal cavity. In right and left nose many polypi, between which pus constantly dripped. Left frontal sinus less transparent than right one. Inflamed remains of tonsils and adenoids. Paracentesis of the drum was done. Pain disappeared and temperature became normal. A foreign colleague had to remove the polypi; a serious secondary hemorrhage with a complicating bronchitis kept patient in bed for weeks. He returned in 1901; the aural inflammation on the right side was cured with cicatrices adherent to the promontorium, but purulent secretion from the left middle ear was now present and purulent secre-

tion of the left conjunctival sac. The nasal polypi had returned; asthmatic oppressiveness and dizziness present. After removal of the remains of the tonsils and the polypi, the pus appeared chiefly to be secreted from the left side. This frontal sinus could be easily probed; the knob of his (SH^5) magnetic sound could be demonstrated 1.5 cm. above the brow. The canula was easily introduced and with the first lavage fetid pus was discharged. The general condition improved markedly, so that against his insistant advice the patient refused any further operation; the secretion from the left ear had stopped, but not from the ethmoid and frontal sinus. He then returned once a year for removal of the obstructing polypi. He went through a serious pneumonia, which weakened his heart; the attacks of headache increased and his general condition grew worse, he became apathetic; rheumatic pains in back and limbs. A year and a half later Struycken was called for serious facial pain and protrusion of the left bulb. The eye was projecting about 1.5 cm., slight chemosis, painful swelling in the inner upper corner of the orbit. Iris-reaction and fundus normal; all ocular motions possible with restriction. Polypi and pus in the nose. Violent pain in the forehead at the left side and around the eye for about six days; temperature was 36.4°. Next morning the left frontal sinus was opened; it was filled with fetid pus and also some accessory cavities behind the brow. The entire anterior and lower walls were removed and a large opening made in the nose by resecting the ethmoid cells. The origin of the protrusion was not found. The following day the temperature was 38°; less pain in the left forehead, but pain in the left upper molars and also the right temporal region. The maxillary sinuses showed less transparency than formerly; after trephining the left one and perforating the septum, which divided this cavity in an anterior and posterior part, cheesy pus was washed from the posterior cavity. This cavity was connected with the ethmoid cells. The second day, temperature was 37.5°; slight bronchitis; much purulent secretion from the ethmoid cells; protrusion slightly diminished; slight general stiffness of the muscular apparatus; euphoria. The third day the temperature was 39°. The entire ethmoid was removed with the medial wall of the orbit; the superior wall of the orbit as far as the fissura orbitalis superior consisted of a series of non-communicating bone cells filled with thick yellow

odorless pus. The sphenoid, which showed no changes, was not opened; the antrum was free from pus. Temperature decreased during the evening to 37.3°, patient felt well without any pain. Next day temperature was again 39°. The protrusion was much diminished, the eye less movable, but nearly insensible to touch; iris-reaction normal; the arteries in the fundus less filled, veins somewhat wider. No bacteria in the blood, many multinuclear leucocytes. No swelling of the spleen. Patient became delirious. The following day the confusion increased, tongue was dry. Respiration slightly of Cheyne-Stokes type. Stiffness of the neck increased. At the right side now and then spontaneous contractions; the general muscular force at that side has decreased. This right side of the body was paralyzed on the following day; coma increased; left pupil wide without reaction. The 8th day after Struycken called the temperature was 39.2°. The left frontal lobe was explored, the dura looked normal, no pus after puncture. On the right side, frontal sinus and ethmoid cells were opened and found filled with pus, without direct communication with the brain cavity. In the afternoon the temperature arose to 41.2° and respiratory paralysis resulted in death. Section showed the convexity of both hemispheres opaque; at the base of the pons some flakes of pus. On section the ramus ophthalmicus nervi trigemini bathed in pus and the vena ophthalmica was closed by a non-purulent thrombus. The nerve, entirely softened by pus, could be followed as far as the brachium cerebri ad pontem, where a focus of the size of a hazelnut was found, the pons itself being narrow. No other abscess formation in the brain. Some small bone cavities were found in the neighborhood of the glandula pinealis and of the fissura orbitalis superior, filled with pus; these last ones, which were not opened during the previous operations, seemed to be in direct communication with the ramus ophthalmicus. Both the sphenoid sinuses were free. The other nerves, as far as visible in the cerebral cavity, were not infiltrated with pus. Probably it would have been better to have opened at the right and left side all bone cavities in one sitting, but patient was afebrile and it was possible that edema of the retrobulbar tissue caused the protrusion. The euphoria and decrease in temperature after the second operation set at ease and did not encourage further manipulation as the opening of the different cavities in the roof of the orbit took much time and exertion. To ex-

pose the dura mater is not at all an indifferent question, when these cavities are infected, as meningitis easily follows. But even the most radical operation would not have succeeded here. *Blaauw.*

Purulent Bone Cyst of the Inferior Turbinate.

JACOB COHN (*Monatsch. f. Ohren.*, February, 1908) reports a case of bone cyst of the inferior turbinal which was probably congenital. It had lately become inflamed, and a fistula formed on the upper surface of the turbinal through which it had discharged. The cyst had no connection with the maxillary or antrum. *Wood.*

Submucous Resection of the Nasal Septum.

GUSTAV KILLIAN (*Beiträge z. Anat., Phys., Path. u. Therapie d. Ohres., der Nase und des Halses,* March 10, 1908) suggests for anesthesia in submucous resection of the nasal septum, a perineural injection of cocain, which he says can be carried out in almost every instance. The nerve which supplies the greater part of the septum is the nasopalatine nerve, from Meckel's ganglion. The front part of the septum is supplied by a branch of the nasal nerve; hence four injections are required, one at the base of each of these nerves or each side of the septum. Killian says that the injection is very easily made except on the convex side in a bad deviation. For making his solution he uses a tablet of adrenalin and cocain. When it is not possible to inject the nasopalatine nerve at its origin on the septum the nerve may be reached further down its course. Ten minutes should elapse after the injection before operation is begun.

In relation to operating on young children, Killian says that with instruments smaller than those used for the adult the operation is fairly easily carried out under general narcosis. He has operated on children as young as four years of age.

Wood.

III.—LARYNX.

The Significance of Tuberculosis Laryngis for the Treatment of Tuberculosis Pulmonum.

PROF. H. BURGER (*Tydschr. v. Gen.*, Feb. 2, 1907) mentions the laryngeal tuberculosis as the most frequent complication

and the most important clinically of the lung tuberculosis. He thinks that it occurs in about 30 per cent of the cases. There is no doubt regarding cure of laryngeal tuberculosis, but there is much difference of opinion as to the number. Nearly always the primary lung affection is of the highest importance for the prognosis; in many cases the amelioration or impairing of the larynx goes on equally with the lungs. A permanent cure can only occur when the tuberculous tissue in both organs is changed into cicatricial tissue. Cure of a larynx tuberculosis without local treatment, only dietetic-climatic, is only very rarely seen. Burger only saw temporary amelioration, but never a spontaneous local cure; but he saw more than once patients return from a climatic or sanatorium cure with very much better lungs, while at the same time a slight laryngeal affection had become worse. A tuberculous larynx has to be treated after the rules of the modern tuberculosis therapy with functional rest, that means to remain silent, and as not less important, the local or surgical treatment must be instituted. This includes application of chemical agents to the tuberculous ulcera, the most important of which is lactic acid, the scraping of ulcerated surfaces, the removal of granulations, the cutting away and burning galvanocaustically of granulations and infiltrates.

Three histories follow, which prove that consequent treatment will give satisfactory results:

1. A clergyman, 44 years old, consulted Burger September 10, 1898, for slight complaints in swallowing and now and then hoarseness, no cough. Some years previous he had a serious lung disease with tubercle bacilli in the sputum. His thorax was flat, respiratory sounds in both apices weak, but no rhonchi. Laryngoscopically an irregular ulcerative swelling at the posterior wall of the larynx.

Treatment.—Four times, application of 10 per cent carbolglycerin (October 30-November 3, 1898), cutting away of the swelling with the double curette (November 4) and 30 times, application of 30 per cent carbolsulforicinol (November 20-December 20, 1898). This treatment did not produce any reaction. At the end the posterior wall had entirely cicatrized. The cut away pieces contained tuberculous tissue with cheesy tubercles. On July 30, 1900, a small raised point is visible, which is cauterized for security sake. July 17, 1901, acute laryngitis with red vocal cords and light swelling of the posterior wall.

2. A 22-year-old nurse began treatment in December, 1895. She began suffering four years previous with cough; spat blood twice, one and five years ago, sometimes pain in throat, increasing hoarseness. She was weak, had night sweats, was emaciated and had pains in back and thorax. Aphonia through ulceration of the left vocal cord with granulations, also the anterior half of the right vocal cord, while the under side of both cords was much swollen. The posterior wall was free. Infiltration of apex of the left lung and tubercle bacilli in the sputum. The treatment, while in the hospital, was difficult through lack of appetite, tendency to vomiting, hypersensitiveness of the larynx, insufficient action of cocain and the additional hysterical temperament of the patient. Besides general measures, the larynx was frequently treated galvanocaustically and with pure lactic acid; the treatment lasted for about a year. The larynx looked then normal except a slight redness and swelling at the posterior part of the left vocal cord. During 1897-1902 a few circumscribed recurrences appeared, always at the posterior part of the left vocal cord, which healed with pure lactic acid. The last treatment was given in May, 1902; two galvanocaustic applications. Since the larynx has remained normal, the apex of the lung has cicatrized. In 1904 and 1906 she went through a full term pregnancy, both times the danger spot at the posterior end of the left vocal cord showed a slight redness with very slight swelling without any other disturbance.

3. A 22-year-old, hired girl, from a tuberculous family, came in April, 1895, under treatment for tubercular infiltration of the apex of the right lung, a scrophuloderma of the left cheek and a high degree of laryngeal tuberculosis; ulcerating infiltration with granulations of the entire right half, the vocal cord could not be discerned, the posterior wall was moderately thickened. The treatment consisted in removal of the new formation and applications of pure lactic acid, which was often disturbed by new formation of the removed granulations. The last curettement was done November, 1895, and the first of December the patient was discharged. The applications with lactic acid were continued till February, 1896. Since then no treatment. The larynx looks normal, so that it is even impossible to recognize the previously diseased side; the lung also remained cicatrized.

These cures of 8, 10 (4) and 11 years' standing illustrate the

possibility often doubted of lasting success in laryngeal tuberculosis. The last two cases are examples of very important tuberculous affections, with very dubious prognosis. After discharge from the hospital both patients took up again their hard work. The voice regained in all three cases its normal sound. To make the focus in the larynx harmless will have a supporting influence for the cure of the lung condition.

The surgical treatment demands not only a complete technic, but also a large amount of patience, perseverance, faith. Who retires to the camp of the nihilists, disheartened by a dozen disappointments and failures, cannot judge fairly regarding the efficiency of this treatment. Admission to this sanctuary only is allowed the faithful. Another cause of failure is the difficulty and still the necessity of a long lasting treatment and control during months and years.

High altitude is not unfavorable for laryngeal tuberculosis. But a patient with laryngeal and lung tuberculosis should not be taken in any sanatorium, unless technically well trained laryngological help can be given. The family physician never should send a tuberculous patient away without an examination by a competent laryngologist for detection of a latent laryngeal tuberculosis. *Blaauw.*

IV.—PHARYNX.

Something About Hemorrhage After Tonsillotomy and a New Method of Relief.

I. C. HENKES (*Tydschr. v. Geneesk*, January 26, 1907) describes first the different anatomic possibilites, which may lead to a hemorrhage and all the usual ways of checking. He then describes his method, which is based on principle of Heerman and Escat, who bring the posterior arc in contact as much as possible with the anterior by means of a strong silk suture. Henkes had made clips, similar to those used in surgery, but larger and of thicker metal. The distance of both sharp points is about two centimeters. Forceps were made for depositing the clips at their place; its tube can easily be turned 180 degrees, so that it can be used for right and left side. Another forceps was constructed for removal of the clips, so bent that the hand does not obstruct the view; its ends are bent over a length of about 1 cm., flattened in the sagittal direction, so that this part, when the forceps are closed, can be easily pushed

between the outer side of the palatal arches and the inner side of the closed clip. Henkes used this method in a case of a boy, 16 years old, from whom he removed both fibrous tonsils with the tonsillotome. The hemorrhage stopped on the left side spontaneously after a few minutes and on the other side after finger pressure during half an hour. In opposition to advice he took solid food the next day, which produced a pretty strong parenchymatous hemorrhage between the palatal arches. Every method failed to check it, until two clips were introduced 1 cm. lower than the first, a silk thread around each was glued with adhesive plaster to the cheek. The manipulation lasted about five minutes. The slight pain disappeared slowly. An iodoform gauze tampon was before put over the wound; this was removed with the clips after 24 hours. No recurrence. *Blaauw.*

Treatment of Peritonsillar Abscess.

MOLL (*Tydschr. v. Geneesk.*, October 13, 1906). The treatment is incision if an abscess is present, or otherwise a Priessnitz-bandage and wait till an abscess has formed. Moritz Schmidt advised incision even if no clearly defined abscess is present. This is not advisable for the general physician. The pus shows itself first in the fossa supratonsillaris behind the anterior pillar. When the patient puts his tongue out and sounds "e" the space between the pillars may be seen when the cheek is stretched. A strongly bent sound may penetrate behind the anterior pillar after cocainization and nearly always pus is found.

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